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EDITED BY

E. C. SEGUIN, M.D.

S'il est possible de perfectionner l'espèce humaine, c'est dans la médecine qu'il faut en chercher les moyens.

—DESCARTES

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Original Articles.

CROUPOUS PNEUMONIA, AN ACUTE INFECTIOUS DISEASE.*

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HAVING now seen in what manner acute croupous pneumonia differs from a purely local phlegmasia or disease, let us now see how it resembles the acute infectious maladies. We have pulled down our old tottering ruin, let us now proceed to build up our new edifice. The term, acute infectious disease, in its modern acceptation, and as we now must understand it, embraces a group of affections dependent upon the introduction into the blood or infection of the system by certain peculiar specific poisons or germs, whether the result of zymotic action, or consisting of low organisms, capable of reproducing themselves to an endless degree, infection being followed by a definite series or group of symptoms peculiar to each member of the class. Upon the fact that we are using the term in its modern acceptation I would lay particular stress. The term infection does not necessarily imply contagion, acute infectious diseases being both contagious and non-contagious.

* Read before the New York Academy of Medicine, March 17, 1881.

(Continued from Vol. v, No. 3, p. 252.)

Under certain conditions, diseases which ordinarily show no contagious properties may become distinctly contagious; while those which have this property only to a mild degree may manifest a peculiar virulence. These conditions are usually found to consist in overcrowding, filth, and the many other unhygienic states. Facts can be advanced showing that, under certain circumstances, at certain times, and in certain places, acute croupous pneumonia possesses this contagious character.

We know that among animals a contagious disease prevails—in fact, it existed in our own State in an epizoötic form last summer,—known as pleuro-pneumonia, the lesions of which are found to correspond with those of croupous pneumonia in man. Morgagni ("The Seats and Causes of Disease." Trans. by Benj. Alexander, M. D. Lond., 1759, vol. i, epist. xxi, art. 26, p. 604) refers to several peculiarly fatal epidemics, which, by others, were looked upon as contagious, but himself disagrees from this. Dr. C. J. B. Williams ("Cyclop. of Pract. Med.," edited by Forbes, Tweedie, and Connolly, vol. iii, Phila., 1845) long ago wrote that "the epidemic occurrence of the disease is clearly proved, and such has been its extent, that a contagious nature has been ascribed to it." Under the title, "Epidemic of Pleuro-Pneumonia in some Ships of the Mediterranean Fleet," Dr. Brayton, R. N., described before the Epidemiological Society of London a series of cases occurring under his notice, which he considered as contagious, and as resembling the pleuro-pneumonia of animals. Thus, he says (*Med. Times and Gaz.*, January 23, 1864): "Besides several features of resemblance in the symptoms of the pleuro-pneumony in the *St. Jean d'Acre* and *Cressy* to the lung disease in cattle, it is to be noted that there are good grounds for suspecting that the affection was communicated by the sick landed from the vessels to other patients in Malta Hospital." That

overcrowding, bad ventilation, etc., were the great etiological factors in this outbreak, the following quotation strikingly proves: "As to the chief cause of this enormous disproportion in the sickness and mortality, etc., in two ships of the same fleet and similarly exposed, it was clearly shown that this lay in the excessive overcrowding of the men at night in the *St. Jean d'Acre* on the lower deck, while in the *Marlborough* the men were more distributed on the different decks, and greater attention was paid to ventilation of the between-decks. Only fourteen inches space was allowed to each hammock in the former ship; and so thoroughly was fresh, cool air excluded from the men while asleep, that the air above the hammocks was found to be from eight to ten degrees hotter than the air below the hammocks, and so offensively impure as to cause nausea to any one going down from the open air." Dr. Ira Russell ("U. S. Sanitary Commission Memoirs," medical volume, 1867, pp. 319–334) records an epidemic of pneumonia, occurring among six colored regiments and a number of refugees, in 1864, at Benton Barracks, Mo., 784 cases with 156 deaths, from January 1st to May 1st, of which he says (p. 322): "The effect of an epidemic influence is shown by the fact that physicians and nurses who had not been greatly exposed to the vicissitudes of the weather, and the other causes mentioned, have suffered from it. Besides, the surgeons on duty with the regiments in the barracks report that men occupying the same bunks with those affected were very much more liable to be attacked than those more remote. Some of the most intelligent surgeons were led to believe that the disease was actually contagious." In the *Lancet* for September 18, 1875, p. 416, Dr. A. Wynter Blyth reports a number of cases of pneumonia as of a contagious form. He says: "When I came into North of Devon, Dr. Christian Budd, of North Tawton, called my

attention to a form of pneumonia which he asserted was infectious (contagious), an opinion which he formed no less than twenty years ago,—and he related many striking instances in support of his assertion." The following are several of the cases mentioned by Dr. Blyth: A farmer affected with acute pneumonia was nursed during his illness by his niece. This niece became affected with the same disease, and carried it to her husband. A man became ill of pneumonia in April, and died after ten days' sickness. His wife caught the disease, her first symptoms appearing three days after his death. About the same date a farmer's daughter, living a mile from the house of the former patient, became ill of it, and five other cases followed, all in the same parish, consisting of a small village and a few scattered houses (population, 470). A farmer in another parish became ill April 16th, and died on the 18th. The servant-woman went home ill of the same disease about a week afterward, and gave it to her married sister with whom she was staying. Drs. Grimshaw and Moore, as the result of their experience in an epidemic in Dublin during 1874 (*Dublin Med. Jour.*, vol. i, 1874, p. 399, *et seq.*) of what they term "pythogenic pneumonia," conclude that "the bibliography of pneumonia indicates the existence of a form of the disease which arises under miasmatic influences, and is contagious." Dr. W. B. Rodman (*Amer. Jour. of Med. Sci.*, January, 1876, p. 76. *et seq.*) reports a similar endemic of pythogenic pneumonia, which occurred in the Kentucky State Prison, 75 cases in all, which he considers as belonging to the class of miasmatic-contagious diseases of Liebermeister, and which he thinks was contagious; while Dr. Samuel E. James records a second endemic in the same prison, the result of overcrowding (*Amer. Jour. of Med. Sci.*, vol. 74, n. s. 1877, p. 54, *et seq.*). The following extract, taken from the *N. Y. Tribune*, January 21, 1880, refers to the same prison, and relates a condition of affairs which has prevailed in it for years.

CINCINNATI, January 20.—A dispatch from Frankfort, Kentucky, gives the report of the Prison Sanitary Committee, made yesterday to the Kentucky Legislature, on the condition of the Penitentiary. It describes a deplorable state of affairs. The committee says that there are eight convicts confined there who cannot live longer than a few months. There are at least fifty others, some of whom are confined to their beds, who, in all probability, cannot live longer than the latter part of next spring, and about 200 others who are in a state of debility and weakness practically unfitting them for duty or work. The rest of the convicts do not present a healthy appearance, and seem affected by the injurious influences which have prostrated the others. The causes of this state of affairs are found by the committee to be numerous. The penitentiary grounds are badly drained, and the sewerage is so defective that in damp weather water stands in portions of the inclosure, from which arises a malaria rendering the air impure. The yards, cells, and workshops are overcrowded, and the accumulation of filth and general lack of cleanliness within the prison contribute to the generation of disease. There is a general lack of ventilation in the houses and cells. The committee expresses the opinion that one of the direct causes of the unhealthiness of the convicts is due to their not being supplied with a sufficient variety of wholesome food.

The following are some additional examples which I extract from a report by Dr. H. J. Hardwicke, of Sheffield, (*Gaz, Méd. de Paris*, 1876, ii, p. 515): A minister suffering from acute pneumonia was nursed by one of his relatives during his illness, who was attacked by the same disease, and in turn communicated it to another relative.

An old man, upon the point of death from this same malady, sent for several of his kindred in order to see them for the last time. Each of them was in turn attacked by the disease. The latest epidemic of pneumonia reported as contagious is by Dr. Adolf Kühn (*Deutsches Archiv für Klin. Med.*, Bd. xxi, 1878, s. 348, *et seq.*). It was, he thinks, distinctly contagious; the attendants during its prevalence in the prison were affected, as also the chronically sick; and the disease was conveyed by visitors to other persons

who did not come near the prison or its inmates. In the *Berl. Klin. Wochschr.*, No. 37, Sept. 15, 1879, p. 552 *et seq.*, he further writes: "It is positive that the epidemic form of pneumonia occurring at certain times and places bears the distinct characteristics of a specific infectious disease." Barella ("Note sur la Pneumonie miasmatique ou zymotique," *Bulletin de l'Acad. de Méd. de Belgique*, No. 2, 1877) studies, under the name miasmatic or zymotic pneumonia, a disease occurring, more especially during the summer, in an epidemic form under conditions of bad hygiene, crowding, insufficient ventilation, deleterious miasms, and contagion.

The characteristics of acute infectious diseases are:

1st. The varying frequency of their occurrence as regards time and number affected, some years being marked by their absence or the small numbers attacked; while at others raging as an epidemic, that is attacking numerous individuals simultaneously or successively.

One of the most remarkable facts in the history of the acute infectious diseases, is their varying frequency, sometimes appearing as sporadic cases only, at others prevailing through a wide circle of territory. Usually we meet with cases of most members of this class at all times, here and there; but, suddenly and frequently, without any assignable cause, cases multiply, an epidemic is prevailing. So it is with measles and scarlatina, with diphtheria and typhoid or typhus fever, with whooping-cough and parotitis, and so on down the list. It is a noticeable and well-known fact that during certain years cases of pneumonia are rare, occurring only as single cases, here and there, sporadic; whereas at other times patient after patient is met with, until, not infrequently, we find ourselves dealing with a veritable epidemic.

Such epidemics of pneumonia have been described even since the sixteenth century (Haeser, *Geschichte der Medicin*,

Bd. ii, s. 344, 1865). Among the epidemics recorded by earlier writers, I have already referred to several by Morgagni. Among modern epidemics we have those already mentioned as contagious. Many simple epidemics have been reported by modern writers, but it will suffice here to merely mention a few. Diehl (*Virchow's Jahresb.*, 1868, Bd. ii, p. 95) has twice observed an epidemic in the prison of Christiana, the first in 1847, the second in 1866-67. Couldrey (*The Lancet*, vol. ii, 1878, p. 701,) reports a local epidemic of pneumonia prevailing during the month of May at Scunthorpe, occurring in two small streets, the sanitary surroundings of which were bad. "There were ten cases. Febrile symptoms preceded the pneumonia three and sometimes four days. Diarrhœa was present in two cases, abdominal tenderness in every case. A well-marked crisis happened on the eighth or ninth day, the temperature falling below normal. One case proved fatal. Fine crepitation was first detected on the evening of the third day, slight dulness on percussion on the morning of the fourth day, and then followed the usual signs of pneumonia. There was also obstinate diarrhœa, prune-juice-colored expectoration, and great prostration. Death took place on the eighth day."

Dr. Henry H. Smith (*Phila. Med. Times*, vol. ix, 1879), in a discussion before the Phila. County Med. Soc., remarked that owing to some unexplained cause deaths from pneumonia had multiplied to an extraordinary degree, one-eighth of the whole number of deaths in Philadelphia being from this complaint. The large mortality may, he thinks, be due to the fact "that there is an epidemic." Cullen ("First Lines of the Pract. of Physic," N. Y., 1793, vol. i, p. 188) states "that the pneumonic inflammation has been sometimes so much an epidemic, as to occasion a suspicion of its depending upon a specific contagion, but

I have not met with any evidence in proof of this." Lebert has convinced himself of its epidemic occurrence in Switzerland; Griesinger affirms that in malarious districts it has a tendency to assume an epidemic character; and Prof. Flint, Sr., states that it has been known at certain times and in certain situations in the Southern States to prevail to an extent entitling it to be called an epidemic. Hirsch was able to collect the records of 163 epidemics, prevailing in various parts of the world.

Pneumonia, like several of the members of the infectious class, prevails at times with other diseases of the same group, as with measles; while there is marked and remarkable coincidence between typhoid and pneumonia years. Sometimes, again, pneumonia occurs in an individual conjointly with an infectious disease, as typhoid fever, measles, scarlatina, etc. Here we must remember that it is not at all uncommon to meet with patients suffering at the same time from scarlatina and diphtheria, or measles and whooping-cough, etc., and that there is no rule governing the infectious diseases which indicates the impossibility of two of them occurring conjointly in the same person. However, it must be stated that coëxistence of acute croupous pneumonia in the same individual with some one of the infectious diseases is comparatively rare.

The laws governing epidemics have yet to be accurately determined; those known being principally related to overcrowding, imperfect ventilation, filth, neglect, and certain atmospheric and telluric influences. Epidemics of pneumonia have usually existed just where such influences prevail, as in cloisters, prisons, barracks, etc. Those referred to by Morgagni occurred among nuns; those of Rodman, Kuhn, Diehl, and others, in over-filled prisons; that of Russell, in barracks, ochlesis exerting in this instance a marked influence; that of Brayton, on board over-crowded ships-of-

war; and that of Couldrey, in two streets in a bad sanitary condition. We sometimes meet with instances of so-called "abortive pneumonia," that is, where all the symptoms of an acute pneumonia terminate in restoration to health in from 32 to 74 hours. Juergensen, Flint, Wunderlich, Bernheim, Leube, and others, have seen such cases. Prof. Leube describes (*Allegemeine Medic. Central-Zeitung*, 1877, No. 34) two cases of so-called transitory pneumonia, which he considers merely abortive forms "of this infection in individuals who have a resisting power against the special poison" (Dobell, "Annual Reports on Diseases of the Chest," vol. iii, 1877, p. 399). Now, in epidemics of such diseases as typhoid and typhus fevers, as is well known, we are constantly meeting with abortive cases. The same is true of other members of the class. Epidemics of the acute infectious diseases are often characterized by distinct varieties, or variations in their clinical aspect, especially as regards mild and malignant forms. The same is true of pneumonia,—witness some of the modifying names employed, as, for instance, the typhoid, bilious, malignant, asthenic, contagious, etc., forms.

2d. A second characteristic of acute infectious maladies is inability to produce them experimentally, except artificial propagation, by the employment of the specific poison of the special disease, be considered such. We have already seen in an earlier part of this paper, that failure has uniformly followed attempts at artificial production of the disease under consideration.

3d. A stage of incubation. The incubative stage of many of the infectious diseases is, as yet, entirely unknown; I can only state that in the epidemics of contagious pneumonia recorded, an uncertain interval elapsed between the time of supposed exposure and the appearance of the first symptom of the malady: in one instance, three days after

the death of the infecting patient; in another, in about a week; and in several others, at indefinitely mentioned periods. Ritter (*Deutsches Archiv für Klin. Med*, Bd. xxv, 1879, s. 52, *et seq*) found this stage in one series of cases, five in number, lasting from nine to thirteen days; while in another group of two cases, the period of incubation was about from 21 to 24 days. According to Traube, the contagious form of the disease has a stage of incubation of three days. Of course such a question can only be decided from the study of a large number of appropriate cases, such as at present I find impossible to obtain.

4th. An initiatory or premonitory stage. That in many cases of pneumonia there is a prodromal stage, lasting for from a few days to several weeks, as a series of vague, indefinite symptoms, we have already seen.

5th. Uniform or classical course, undeviating sequence of symptoms, except such as are modified by or due to the special epidemic influence prevailing. There is no other disease, so far as I am aware, which has so definite and classical a course as acute lobar pneumonia. Usually beginning abruptly with a chill, followed for a determinate period by definite, well-marked, and almost unvarying symptoms, it terminates, if in recovery, by a sudden and almost plunging crisis, a sudden disappearance of all constitutional manifestations. Local symptoms still prevail, but so do they in typhoid fever, diphtheria, etc., during convalescence; the restoration to a normal state of the affected tissues being itself only gradual, is necessarily attended by a gradual disappearance of symptoms referrible thereto.

As is well known, during different years variations in the intensity or predominance of certain phenomena have been noted in infectious troubles, as also in the type of the disease. Such is also true of pneumonia. For instance, the

occurrence of herpes labialis during certain seasons, and its absence at others; the presence of marked gastric symptoms, or the appearance of icterus, giving rise to what has been known as the icteric or bilious form; the prevalence of the so-called asthenic or malignant variety, marked by symptoms of great prostration, stupor, or delirium; or the occurrence of diarrhœa, as has been noticed in certain epidemics.

6th. Another characteristic of infectious complaints is absence of direct relation between constitutional symptoms and visceral lesions. I have already dwelt upon this point, and will only here quote the following from Juergensen (Ziemssen's "Cyclop. of the Pract. of Med.," vol. v, p. 146): "Small consolidations with high fever and severe constitutional symptoms, and solid infiltrations with a comparatively slight fever and general disturbance, this is the rule and not the exception."

The fact that extension of the disease is attended by increase of symptoms is not contrary to the laws governing infectious diseases; for, do we not note the same in extension of the diphtheritic and erysipelatous processes?

7th. Occurrence of certain complications in certain epidemics. At times pneumonia is marked by entire absence of complications, while at others a large percentage of the patients suffer from them. Thus, during my first winter in Bellevue Hospital, most of the cases were simple and ran a mild course; whereas, in my second winter, among 24 cases coming under my notice, 9 were attended by complications directly dependent upon the pneumonia: three with pleurisy, one of which was acute and diffused, two subacute with serous effusion; two with pericarditis; one with acute peritonitis; one with general bronchitis, this case being also attended by abortion; one with acute empyema, endo- and pericarditis, and verticular meningitis; and a ninth with gastro-

duodenitis, icterus, cholæmia, and acute parenchymatous nephritis. The prevalence of complications is thus referred to by Wilson Fox (*loc. cit.*, p. 677): "The frequent association of albuminuria with pneumonia can scarcely be regarded as a mere accidental complication, and it is by no means improbable that the kidneys are, under these circumstances, implicated by the same cause as the lung. Other glands also occasionally suffer, as the parotid gland; gastro-dudodenal catarrh and some degree of affection of the liver are also complications. In addition to these, the serous membranes tend also to become implicated as part of the primary disease, and when these relations of pneumonia are regarded as a whole, it appears that those organs are most likely to suffer which are most commonly affected by recognizable conditions of blood-poisoning." If we examine the statistics quoted by Juergensen, we will note that different places are marked by different rates of occurrence of certain complications. Thus, pleurisy with effusion complicated 5.2 per cent. of the Vienna cases, 4 per cent. of the Stockholm cases, and 15.3 per cent. of the Basle cases; pericarditis, 0.5 per cent. in Vienna, 0.9 per cent. in Stockholm, 3.09 per cent. in Basle; endocarditis, 0.2 per cent. in Vienna and Stockholm, 0.9 per cent. in Basle; meningitis, 0.1 per cent. in Vienna and Stockholm, 1.3 per cent. in Basle. Parotitis has, in a few patients, been observed by Béhier and Fox, such cases being particularly fatal, in these respects resembling typhoid fever in which a similar condition has been known to occur. Speaking generally, it may be said that each member of the infectious group of diseases has a special class of complications, that of pneumonia being its tendency to involve the serous membranes, as will be observed from the foregoing.

8th. Self-limitation. Thus, typhus fever runs its course in about 14 days, typhoid in about 28 days, pertussis in

some 6 weeks, measles in 7 days, and so on through the list. To no other disease, I will venture to assert, can the term self-limited be applied with greater justice than to pneumonia. Of course, as in some acute infectious diseases, we find in a small percentage of cases variations from this rule, but such cases are comparatively rare. Of 867 cases, terminating by crisis, reported by various authors, 677 ended by the eighth day, and all by the eighteenth.

9th. A rate of mortality varying with each epidemic. Statistics show that the rate of mortality in pneumonia varies from 2 per cent. to $3\dot{3}$ per cent. (Andral), the death-rate differing greatly in different years, even under the same methods of treatment. Thus Huss' statistics show rates ranging from 9.1 per cent. to 14.1 per cent. under antiphlogistic measures, and from 6.1 per cent. to 13.4 per cent. after the abandonment of this plan. Brandes' mortality was, one year, 5.4 per cent., and the following year, 31 per cent. Of my 24 cases 12 died. Fourteen were complicated; of these 11 succumbed. Of the simple cases only one died, this being the old woman 72 years of age, who, while suffering from great mental depression, made an attempt at suicide by drowning. The cases of the previous year had been marked by their great mildness, but few terminating fatally.

10th. Localization of morbid changes to some organ or sets of organs. Typhoid fever seizes upon the solitary and agminated glands of the small intestines, diphtheria involves the pharynx, mumps the salivary glands, and so of other members of the class; the specific poison of each disease seeming to have a selective power toward certain organs or sets of organs. The consolidation of the lung tissue, therefore, in pneumonia, may be considered the essential morbid lesion, in the same sense that ulceration of the solitary glands and Peyer's patches is the essential lesion of typhoid fever. In blood diseases it is a well-

recognized fact that the blood poisons produce their most marked effects on glandular organs; it is in these that the local manifestations of the constitutional disease show themselves. The lungs are closely allied to these glandular organs: first, by their anatomical structure; second, by their important functions as purifying agents of the blood; and third, by their great and almost ceaseless activity. In this resemblance, therefore, we may perhaps find a partial explanation for their involvement by the causative poison of pneumonia. The fact that the disease may primarily attack a single lobe, finally extending to the other parts of the lung, is not contrary to this theory; for, do we not see the same thing modifying the lesions in typhoid fever, mumps, diphtheria, etc. In typhoid fever the ulcerative process may involve only a few of the lymphatic glands of the small intestine, or may be very much more extensive, as in a case I have seen, where the ulcerations not only extended throughout the greater part of the small intestine, cœcum, and lower part of the ascending colon, but even into the vermiform appendix, two small ulcers being found in this situation. Parotitis usually is at first single, but tends to become double. The diphtheritic process may at first localize itself in the pharynx, and from there may advance into the nasal passages or the larynx, while the exanthem of the eruptive diseases may be very scanty or very abundant.

11th. Uselessness of remedies against the disease itself, treatment being almost entirely symptomatic. In acute infectious diseases we do not treat the disease itself, but rather such of its symptoms as may demand attention. The same holds true of pneumonia; fever is combatted by antipyretics; pain, cough, sleeplessness, etc., by sedatives; exhaustion or heart failure, by stimulants. The old method had for its object the cutting short of the disease; our

modern method ignores the lung disease itself, but turns its attention to the avoidance and preventing of intercurrent dangers. "If," says C. Handfield Jones (*Med. Times and Gaz.*, vol. ii, 1873, p. 118), "then, pneumonia, as we see it now-a-days, is a fever and not an inflammation, it is clear that the object of treatment must rationally be, not to arrest it, but to conduct it to a safe termination. This view is very generally acted on, and is strongly supported by Dr. Bennett's experience, who makes it his aim to conserve the patient's strength."

12th. The great characteristic of acute infectious diseases is their specificness; "under all circumstances, a given kind of disease is solely due to a given kind of morbid agent or cause" (Liebermeister, Ziemssen's "Cycloped. of the Pract. of Med.," vol. i, p. 14). No matter what other conditions may be present, the producing element for each member of the class is always a special, infectious principle, a specific poison acting on and through the blood.

The theory that pneumonia is due to a specific blood poison is not a new one. As long ago as the time of Morgagni, physicians entertained the suspicion that the disease might depend upon a blood poison. Among others of the earlier writers, we have Carolus Strackius ("Nova Theoria," Morgunt, 1786), who declares himself positively in favor of a miasmatic cause for the malady. Cullen, while advancing no arguments against the belief of the causation of the disease by a specific poison, is only able to state that he has met with no evidence in proof of this (*loc. cit.*, p. 188). J. Frank, Skoda, Robert Latour, Marrotte, were believers, to a certain extent, in the miasmatic origin of the disease. Laennec ("A Treat. on the Dis. of the Chest and on Mediate Auscultation," 3d ed., trans. by John Forbes, N. Y., 1830, p. 225) remarks: "It is possible that the epidemic peripneumony, is often owing to an analogous cause, that is to

say, to deleterious miasms, which have entered the system by means of the cutaneous or pulmonary absorbents, since nothing is more common than to meet with cases of this disease, to which we can assign no occasional cause. How many persons are seized with it, in their very chambers, and in spite of the utmost care taken of their health." Huxham and Fr. Hoffmann considered the disease a fever, of which the pulmonary changes constituted merely the principal localization. Pons, according to Leichtenstern, affirmed that "pneumonia is a general disease, complicated by pulmonary inflammation," while, according to the same writer, Trousseau maintained that in this disease "the blood contains another morbid element, of the nature of which we are ignorant, but whose existence is revealed to us by constant morbid manifestations," viz., pneumonic inflammation. In 1860 Dr. Parkes (*Med. Times and Gaz.*, vol. i, p. 186) wrote that "it (pneumonia) is a blood disease of some sort, consisting, in part, in an augmentation of the fibrin in the blood, as in acute rheumatism." We know now that the excess of fibrin in the blood, here referred to, is a consequence, and not a cause of the disease (Virchow). Dr. Dupré, in an article on catarrhal fever, written many years ago, speaks of this infectious theory of pneumonia (Hallopeau). In 1866 Prof. Wm. H. Draper, in a discussion before the N. Y. Academy of Medicine, affirmed that "if it be true that the lesion is a sequence, in point of time, of the pyrexia, then it is altogether probable that it is a secondary phenomenon, and a necessary and conservative process, by which a blood poison is eliminated from the circulation. It is true that chemistry has not yet discovered any specific poison in the blood of persons suffering from pneumonia; but we are not without strong presumptive evidence in favor of this theory. These considerations certainly lend support to the theory that pneumonia is something more than a

local disease, and is rather an essential fever, having a characteristic lesion, like small-pox or scarlet fever (*Bull. of the N. Y. Acad. of Med.*, vol. ii, 1866, p. 519). In an article, entitled "Note sur la Fièvre Herpetique," Parrot, in 1871 (*Gaz. Hebdom.*, 14 juillet, p. 374, et 28 juillet, p. 412, 1871), makes the following remarks (p. 416): "In a word, does the anatomical lesion govern the disease; or, conversely, is the principal rôle played by the fever? Seeing the impossibility, at present, of answering these difficult questions, it has appeared to me appropriate to point out the analogy existing between 'herpetic fever' and acute pneumonia, as shown by our observations." Juergensen is the great modern exponent of the infectious theory of pneumonia. Among others who have announced their adherence to this theory, I will mention Prof. Austin Flint, Sr. (Tr. of the Med. Soc. of the State of N. Y., 1877); Dr. Moellmann (*Berliner Klin. Wochenschr.*, No. 12, 1879); Dr. Henry H. Smith (*Phila. Med. Times*, vol. ii, 1879); Dr. James Andrews (*Med. News and Lib.*, Sept., 1877); Bernheim (*Gaz. des Hosp.*, 1877, p. 228); Marrotte (*Arch. gén de Méd.*, 1873); O. Leichtenstern (*Volkmann's Sam. Klin. Vort.*, No. 82); and Cohnheim (*Vorlesungen über allegemeine Pathologie*, Bd. i, 250, Berlin, 1877). Friedreich (*Volkmann's Sam. Klin. Vort.*, No. 75) goes so far as to admit the occurrence of a type of pneumonia which is infectious, but does not speak in the same way of acute pneumonia in general; while O. Sturges ("On Pneumonia: its Natural History," etc., Lond., 1876) considers the disease neither a local inflammatory nor general one, but rather one lying midway between the two. Cohnheim classes the disease among the miasmatic-contagious, and maintains that, without being directly transmissible, it is never in any locality developed without having been previously imported (Hallopeau).

Some of the names used formerly to designate the dis-

ease would seem to imply a specific cause. Thus, we have the term, febris pneumonica (Hoffmann). But here we must remember the carelessness and inappropriateness with which such designations were often formerly employed; and the fact that inflammation of the lungs, peripneumonia, etc., were used as terms synonymous of the same. The like is true of the French "fièvre pneumonique," and the "lung fever" of our laity. Dr. Flint proposes for the disease the name "pneumonic fever."

The question now arises, pneumonia being an acute infectious malady, how does its specific poison gain entrance into the system, how is it taken into the body? We know that the poison of typhoid fever is taken into the stomach in drinking-water, contaminated milk, and the like, and it is presumed that it enters the system by absorption through the solitary and agminated lymphatic glands of the intestines, on which it produces its most marked visible effects. May it not be that the germ or poison of pneumonic fever enters the body through the lungs by inhalation, and then follows a course analogous to that of the typhoid germ? In support of this doctrine we have the following: According to Dr. Parkes, the poison of the contagious pleuro-pneumonia of animals is contained in the exudation, probably the epithelium and pus, which, taken into the lungs of a healthy animal, will reproduce itself and give rise to the disease. "Considering," he says, "that the pleuro-pneumonia of cattle is probably propagated through the pus and epithelium cells of the sputa passing into the air-cells of other cattle; that even in man there is some evidence of a pneumonia or phthisical disease being contagious" ("A Manual of Practical Hygiene," p. 74, 1864). Barella (*Gaz. Hebdom. de Méd. et de Chirurg.*, 2 mars, 1877, p. 136) advances the following: "The typhogenic miasm may enter the economy by two avenues: the

digestive mucous membrane, and the respiratory mucous membrane; if it seizes primarily upon the digestive passages, it produces typhoid fever; if it acts preferably upon the respiratory mucous membrane, it gives rise to *typhoid, miasmatic*, or *zymotic pneumonia.* The frequency of pneumonia during certain epidemics of typhoid fever has been noticed, and all practitioners are aware that at the beginning it is sometimes very difficult to differentiate the one from the other." Klebs (*Archiv für Experiment. Path. u. Pharm.*, Bd. iv, 1875) affirms that he has been able to determine the nature of the infectious agent. He describes a "monas pulmonale," inoculation of which in animals was, he claims, followed by the development of the malady in question, also stating that O. Weber had already shown, experimentally, that the fluid obtained from pneumonic lungs possessed pyretogenic characters to a high degree; and Kuntze, following this hypothesis to its extremest consequences, advises and puts into practice, with the object of destroying the infecting germ, the treatment of pneumonia by subcutaneous injections of carbolic acid.

Moore and Grimshaw relate that in a certain high-school, following the placing of a ventilator by the sewer authorities, the institution became infected by sewer-gas. Shortly thereafter, cases of pneumonia began to develop, and, as a consequence, the school had to be closed. The ventilator was removed, and the cases of pneumonia ceased to occur. For many years previous to this outbreak, there had been no sickness in the institution. The inference is here certainly very strong that the sewer-gas and pneumonia stood in the relation to each other of cause and effect. If such be the case, the avenue by which the poison gained access to the system is obvious, viz., by inhalation.

Finally, quoting from Wilson Fox (*loc. cit.*, p. 677): "Whether the blood poison is eliminated by the exudation

process must remain a matter of hypothesis, though by the sudden cessation of the pyrexia when this stage has advanced to a certain degree would appear to lend some support to this view, and particularly when we remember the analogy, and even the various phases of transition which exist between exudative and secretory process." However, it remains for future research to decide this question.

To sum up, acute lobar pneumonia is an acute infectious disease, dependent upon the introduction into the system of a specific poison, the visible expression of whose activity is a croupous inflammation of the lungs, and may be classed among the miasmatic-contagious group, belonging thus to the same class of maladies as typhoid fever. In all probability, the poison is taken into the organism by absorption through the lungs, that is, by inhalation.

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EXPERIMENTAL AND MICROSCOPICAL STUDIES ON THE ORIGIN OF THE BLOOD GLOBULES.

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THE objects of this paper are to give the result of a repetition of Onimus' experiments on the "origin of the white blood corpuscles," and to place on record an account of an undescribed method of development that is constantly going on in the adenoid tissues. As given by Flint, these experiments of Onimus are as follows:

The serum from quickly-drawn blisters, after having been freed by filtration, etc., etc., from all its organized elements, is placed in bags of gold-beater's skin. These sacks are then placed in the subcutaneous tissues of rabbits, and after a sojourn of two or three days their serum is found to contain a variable number of leucocytes.

His conclusions are that the corpuscles have sprung up *de novo* from the blastema, and by analogy he argues that there is a spontaneous generation going on in serum wherever it is found.

I have repeated these investigations, and in two directions have pushed them further than their author; that is, instead of the blastema, in the course of the experiments I used four different liquids, and in all cases, besides the fluids, I examined the gold-beater's-skin after its removal.

In addition to the serum I used a weak solution of chloride of sodium in water, a mixture of this with the white of an egg, and lastly the clear part of the egg alone. The animals used were cats; the length of experiments from 17 to 50 hours; the thickness of the enclosing membranes was in most instances one, but in two cases two, layers of the gold-beater's skin. In all cases I examined both membrane and blastema before the introduction to the cat, and thus made sure that no organisms were present. My results were that in every case, except where I used a varnished membrane, I found leucocytes in the blastema, and wherever they were found in the liquid the walls of the enclosing bag were sure to be crowded with the same organisms.

The only things that seemed to influence the number of the corpuscles were the condition of the containing membrane and the length of time the sack remained under the skin. If these conditions were the same there were just as many corpuscles in the solution of chloride of sodium, or the egg mixtures, as there were in the serum. In the cases where the skin was doubled after a longer time than was ordinarily employed, a few corpuscles made their appearance in the blastema, a few were found in the inner layer of the bag, whilst the outer one contained a great many.

From these facts we are forced to the conclusion that the corpuscles migrated through the walls of the bags, just as they do to the interior of the cat-gut ligatures that are left in similar conditions.

This, however, is only a negative kind of proof, and for something positive I will ask the reader's attention to my recent study of the so-called adenoid tissue.

It is not necessary here for me to give the histology of the organs that contain this tissue, and to repeat that in the lymph glands it is arranged into lymph follicles, lymph cords, and interfollicular strings; in the alimentary canal

into follicles such as are contained by the tonsil, base of the tongue, pharynx, æsophagus, solitary glands, Peyer's patches, etc., etc.; in the spleen into the ensheathing coats of the arteries, and the so-called Malpighian corpuscles, etc., etc. But for our purpose, all that we need to know is that wherever this tissue may be there is a stream of fluid coming into it on one side, which, after working its way through the sponge-like mass, passes out on the other and eventually empties into the blood.

The two questions to which we will now address ourselves are: Whence comes and what is the function of the adenoid tissue?

All histologists agree that in the animal kingdom we find but four varieties of connective tissue and that they are the myxomatous, the fibrous, the cartilaginous, and the osseous. The myxomatous connective tissue is met with almost exclusively in the earliest stages of development of the embryonal connective tissue, and in transient fœtal organs, such as the umbilical cord and placenta. This tissue appears in two varieties: first, in the shape of a protoplasmic reticulum of greatly varying size, with nuclei at its points of intersection, the meshes of which hold the jelly-like mucoid basis substance (umbilical cord). In the centres of the meshes globular and apparently isolated bodies are seen. The other form consists of a delicate fibrous reticulum, having oblong nuclei at the points of intersection, the meshes being filled with single protoplasmic bodies (so-called decidua cells of the placenta), or with a mucoid basis substance with scanty bodies (derma and mucosa of the embryo in the earliest stages).

Recent researches have proved that this mucoid basis substance is not a structureless mass, but that it is pierced by a living reticulum, which is continuous with a smaller net-work which pervades all protoplasmic formations. As

the fibrous reticulum of myxomatous tissue is a protoplasmic formation, its fibres, too, contain a fine reticulum of living matter, which is also continuous with the fine reticulum of its neighbors. So the basis substance, in either its mucoid or fibrous variety, differs from protoplasm only by a chemically altered substance within the meshes. This substance in the protoplasm is a liquid, in the basis substance a semi-solid, though not strictly glue-yielding mass.

As has been known for a long time, comparatively low powers, when brought to bear on the adenoid tissue, demonstrate the presence of a delicate fibrous reticulum, which at the points of intersection is generally slightly thickened and flattened so as to present a plate-like appearance.

These intersections are sometimes provided with nuclei, and the meshes of the net-work are always filled with lymph corpuscles. Although these corpuscles are so closely packed that they often flatten each other, still each one is generally separated from its neighbors by a narrow, light substance which is probably liquid.

Unless the lymph corpuscles be torn apart by mechanical injuries, such as cutting, washing, etc., etc., they are all connected with each other by extremely delicate, grayish spokes, which traverse the intermediate substance in all directions. A like connection always exists between the lymph corpuscles and the fibrous reticulum nearest to them. Most authors claim that this fibrous reticulum of the adenoid tissue is structureless, and exhibits nuclei only at its points of intersection.

This assertion must be based on Canada balsam specimens, for it makes all minute details fade away. My own specimens, cut from fresh lymph glands, or such as had been preserved in a dilute solution of chromic acid, show a well-marked net-work in the fibrous reticulum both in the unstained and in the carmine specimens.

While we are on this subject of the preparation of specimens, let me say, once for all, that if we hope to see the minute structure of this tissue our sections must be cut from fresh or from chromic acid preparations, for alcohol or water destroys the details. If stained at all, it should be done with carmine, or what is better, the $\frac{1}{2}$-per-cent. of chloride of gold. This last named agent has a peculiar faculty for taking hold of the living matter of the most minute organisms and making it stand out in a very satisfactory manner. Lastly, I would state that glycerine seems to be the only mounting substance now known that will preserve tissues absolutely unchanged.

Reasoning by analogy it seems that we are forced to conclude that adenoid tissue is myxomatous, and, therefore, a remnant of fœtal tissue. We know that the myxomatous tissue is abundant in the embryo, and relatively scarce in the fully developed fœtus. In the adult the vitreous body was considered the only remnant of embryonal myxomatous tissue. To this, however, we should add the adenoid, and thus answer our first question.

To get a better idea of this tissue, let us turn to its most minute anatomy, and for the present we will confine our attention to its framework. As I have already said, in the framework, which looks perfectly homogeneous under a 500, with a 1,200 (immersion) we can readily recognize a delicate reticulum piercing nearly all its fibres and plates. In some places, even without the use of a staining reagent, this net-work is just as plain as in the corpuscles themselves, the only difference being that its meshes are a little wider than those in the globule. But the point to which I wish to draw particular attention is, that the granules, at its points of intersection, vary very much in size. Sometimes where they are seen along the edges of broad fibres, or in the centres of very fine ones, they give it a beaded appear-

ance. At others they are so small that they are just barely appreciable. This inequality in size is most probably due

Lymph ganglion of cat magnified 1200 diameters.
aaa. Myxomatous reticulum exhibiting in its interior a delicate reticulum of living matter.
bbb. Granules of living matter arising from the growth of the intersections of the contained reticulum.
ccc. Granules grown into vacuolized corpuscles, and intermediate stages of development.
ddd. Full-grown nucleated lymph corpuscles.
eee. Mesh of the myxomatous net-work filled with lymph corpuscles of all stages of development.
fff. Fine spoke-like threads connecting the corpuscles with the reticulum lying within the myxomatous framework.

to a growth that is constantly going on in these granules, and our finding different ones at different stages of it.

This process does not stop where the lump of living matter can be called a granule, but it keeps on until it has converted it into what is known as a corpuscle. This is accomplished by the smaller granule increasing until it has become so large that the fibre can no longer contain it without showing a slight bulging at the point where the granule lies. This is what gives the beaded appearance just referred to. But as the bead still grows it protrudes more and more from the free surface of the fibre, until it has the appearance of a small homogeneous yellowish corpuscle sticking to the side of the fibre. The corpuscle is not separated from the fibre in this immature state, but retains a connection in the shape of very delicate grayish spoke-like threads, that can be traced directly to the granules within the fibre. This connection is constant in all the different-sized corpuscles, except the very largest, and in all probability is the route through which the corpuscle draws its nourishment. We can see no differences in these growing corpuscles until they are about three-quarters the size of a red blood globule. Then, however, they seem to be divided into two classes. Whether there are two sets of fibres that produce the different corpuscles, or how else it is done, is more than I can say; but I am sure that at the stage I have indicated, one set become more highly refracting than the other, and take more and more of the characteristics of a red blood globule, which they eventually become. The others, however, follow the course that C. Heitzman has described (Sitzungsber. der Kais Akademie der Wissenschaften, 1873) as the course that the elementary homogeneous granule takes in its development into a higher grade of protoplasm. After they reach the size I have already spoken of, a cavity containing a small amount of liquid forms, then similar excavations show themselves, until only a framework of the living matter is left between the

vacuoles. There are communications established between these cavities, and the framework is transformed into a net-work with thickened points of intersection, which are the granules.

With this view of the development of protoplasm we are better able to understand the meaning of the vacuolized corpuscles that we so often meet with. But the different sizes of the corpuscles, the different numbers of their granules, and the varying conditions of their nuclei and reticula, speak for themselves. They are the different stages through which an original granule of the fine reticulum contained by the fibrous net-work is developed into a full-grown lymph corpuscle.

This is further substantiated by the fact that the connection, already described, between the granule that has just passed to the outside of the fibre and the reticulum within it, is kept up through all sizes and shapes of corpuscles, until the full-grown condition is reached. Then, however, this attachment is severed, and the globule passes away with the lymph stream in which it has been bathed so long. This is true of both sets of corpuscles, and can be shown as well in the young red, as in the white. Thus we add a new proof to the old idea that a red globule is nothing but a mass of protoplasm containing hæmoglobine within its meshes; but for the elaboration of this subject I refer my reader to the researches of L. Elsberg.

The organs that I have used in these investigations are the lymphatic ganglia of man, horse, and cat, the spleen of man and cat, as well as the tonsil and thymus gland of children. The characteristics of the adenoid tissue were found to be the same in all, the principal differences being in the proportion of red to white globules. In the tonsil and lymphatic ganglia, the red are very scanty, though they can be found in most fields; but in the spleen they are far

more frequent. In this organ, like the rest, the corpuscles are formed by the development of the granules of the network within the frame, and not by budding of the endothelial plates, as claimed by some. We are now ready to give the reason for the lymph of the efferent vessels containing so many more corpuscles than that of the afferent, as well as to say where the few red globules that are found in the lymph of the thoracic duct come from. The lymph stream, as it passes through each successive ganglion, carries along an increased number of the fully grown elements that have become detached from the parent fibre, and eventually empties them into this duct, through which they reach the blood.

In answering these questions, we are also giving the function of the adenoid tissue, which is to produce the corpuscular elements of the blood.

It has been known for a long time, that as age advances the adenoid tissue becomes more and more scarce, and that the mucous layers and other organs that were once so rich in it, at extreme old age present scarcely a trace. In reality, the thymus gland may be taken as the type of the whole class. For while their degeneration is by no means so rapid, still they all show a tendency to follow its example. This is most strikingly shown in the history of Peyer's patches, as has been brought out by the study of typhoid fever. From this we would conclude that a young animal is the best subject for the study of the adenoid tissue. This I can testify is the case, for as age advances the granules of the reticulum within the fibres become more scanty, and the reticulum itself is by no means so rich as in the early days of life. Thus we see that we live at the expense of our cytogenic tissue. Should it ever be conclusively proved that the white blood corpuscles share in the formation or repair of the structures of the body, we would then have

the complete chain of their history; for we are now sure that they represent only one stage of a development that is going on as long as life lasts, and I am not inclined to believe that this stage is the highest of the series. The conclusions that I have drawn from these studies are:

1st. We must have more and better proof before we can believe that a lymph corpuscle ever arises from a blastema.

2d. That both red and white blood corpuscles are developed from the granules of the reticulum of living matter within the fibres of all adenoid tissues.

3d. That in different organs there is a difference in the proportion of red to white globules that are produced.

4th. That the adenoid tissue is myxomatous, and, properly speaking, a remnant of fœtal life.

5th. That this tissue is stored-up material, from which the blood corpuscles are made throughout life.

6th. That it is highly probable that the exhaustion of this material plays an important part in senile atrophy, and the other torpid conditions of the aged.

Before closing this paper, I wish to acknowledge the kind assistance rendered me in its preparation by Dr. C. Heitzman of New York, in whose laboratory much of the microscopical work was done.

THERAPEUTIC CONTRIBUTIONS.

III.

ON THE USE OF A FEEBLY ALKALINE WATER AS A VEHICLE FOR THE ADMINISTRATION OF THE IODIDE AND BROMIDE OF POTASSIUM, ETC.

By E. C. SEGUIN, M. D.

ONE hears a great deal in remarks and debates at medical societies and in private consultations of the gastric derangement produced by remedies which are of constant use and of unsurpassed efficacy, viz., the iodide of potassium and the various bromides (more especially the bromides of potassium and sodium). This evil result, or the dread of it, is not infrequently interposed against the free use of these salts in large doses for the relief of serious symptoms.

For example, a patient lies comatose from cerebral syphilis, and when the advice is given to administer 3 or 4 grs. potassium iodide every two or four hours, the attending physician very often expresses his fears that great gastric derangement will result, interfering with the digestion of food. I have known the recovery of such a case placed in the greatest jeopardy by such a dread of the local effect of this remedy.

Again, a patient is allowed to have recurring attacks of epilepsy while using small doses of potassium bromide, whereas by giving larger doses the paroxysms might be in-

definitely suspended. The larger doses are not given partly from a fear of bromism in general, but also, I am convinced from numerous consultations, because it is believed that the bromides cause gastric catarrh.

I am perfectly ready to admit that the salts in question may and do cause gastro-intestinal disorder, but I have very rarely observed this in my practice during the last three years. Having, as I believe, found the means of administering the iodide of potassium and the various alkaline bromides in a harmless way (as regards the digestive organs), I fancy it may be of some utility to give a detailed account of my plan of administration.

This plan includes the almost equally important conditions:

1. The use of a simple aqueous solution of the salt.

2. Its ingestion upon an empty stomach (fifteen or thirty minutes before food).

3. Its very free dilution with an alkaline solution.

I. The importance of employing absolutely simple solutions of certain remedies, especially of the bromic and iodic salts, is being more and more realized by physicians, and the nauseous and, as I believe, indigestible mixtures which were imposed upon the profession by high authorities some twenty years ago, are passing out of use. Certainly, in the case of drugs whose remedial effects are as special and relatively simple as are those of the bromides and iodides, it would seem, *a priori*, that giving them in the shape of an aqueous solution were best. Their efficacy can hardly be increased by the addition of other drugs, and their taste certainly cannot be covered up or neutralized by infusions, syrups, etc. It has been my practice for several years to employ a solution of iodide of potassium made by dissolving equal parts by weight of the salt and of water. Experimenting upon a considerable bulk, it has been found that there is a loss by volume of

one-fifth in mixing the salt and water. In other words, a drop of this solution contains about $\frac{4}{5}$ of a grain, or .05. A patient who takes a dose of one hundred drops of this solution does not in reality receive (as is often erroneously stated) one hundred grains, or 6. of the salt, but only about eighty grains or 5. This difference is of considerable importance in the treatment of cases requiring the maximum doses of iodide. Of this solution I direct that so many drops be given in the dilution to be presently described, about half an hour before meals, or before food.

The bromides I have for some years prescribed upon one general or typical formula, varying the ingredients to suit different cases, but keeping the standard dose the same. This will be at once recognized as of great utility in treating a large number of cases of epilepsy in private and in hospital practice. It is needless to defend the use of a standard formula from the charge of routine practice, because reflection will show that with such a type-formula, the doses for each case can be varied infinitely by subdivision and arrangement of quantities of the solution. This general formula is:

℞

Potassii bromidi,	℥ iss or 45.
Aquæ,	℥ vij or 200 cc.

A teaspoonful contains gr. xv, or 1. of the salt.

Another formula, which I often employ, is:

℞

Ammonii bromidi,	℥ ss or 15.
Potassii bromidi,	℥ i or 30.
Aquæ,	℥ vij or 200 cc.

Of this solution also a teaspoonful contains gr. xv, or 1. of the salts.

All of my anti-epileptic solutions are constructed upon this type: one teaspoonful containing gr. xv, or 1. of the

salts. Perhaps the formulas require some explanations. They are not intended as examples of mathematical accuracy in dosage, such as would avoid an error of one grain. They are constructed for practical use in families, and calculated upon the average capacities of teaspoons. These utensils no doubt vary in capacity, but from my own experiments, and from the testimony of others, medical and non-medical witnesses, I have been led to assume that only about seven teaspoonfuls could be obtained from the ounce of solution. Each of my standard bromide formulas contains, practically, 49 doses, which, divided into the total quantity of salts, yields a quotient of very nearly 15 grains, or I. The translations into the metric system also need a word of explanation. They are corresponding and logically equivalent translations, and not at all literal translations, such as abound in medical books and periodicals—translations absurdly exact, and only serving the purpose of disgusting physicians with the use of the metric system in prescriptions. In rendering ℥i by 30., ℥ss by 15., and ℥vij of liquid by 200 cc., the errors are, I believe, about compensatory in each estimation, and after making allowance for a small increase of bulk by the addition of 45. of salts, reckoning the teaspoonful at a little over 4., we obtain the same number of doses as in our English formula, viz., 49 or 50 doses.[1]

Of these various bromide solutions, I direct one or more teaspoonfuls, properly diluted, to be taken upon an empty stomach.

II. The idea of giving the iodides and bromides on an empty stomach is in no wise new, but is in opposition to what I think is the general practice. Influenced by the (de-

[1] It is somewhat surprising, and to me a matter of gratification, that the formulas which I devised in 1874–5 according to the English system, *when I had no thought of employing the metric system*, should have happened to embrace correct metric proportions.

lusive) notion that iodides and bromides produce gastric irritation, most practitioners give them after eating, when they probably undergo more rapid decomposition, and interfere with the process of digestion.

Deposited into an empty stomach, which in normal conditions presents a neutral or alkaline reaction, more especially if guarded by an alkaline liquid, it is a practical reality that these salts are very efficacious, and that they cause no gastric irritation. Theoretically it is almost demonstrable that they are absorbed quickly, fully, and with little if any change. The contact of the solution may act as a solid body or a portion of aliment, and cause an outpouring of acid gastric juice; this is, however, not proven, and if such an event does occur, the acidity thus produced will be antagonized by the alkaline salts of the solution.

III. While serving as resident physician in the New York Hospital in 1865–7, I noted the addition of potassium bicarbonate to prescriptions for potassium iodide by some of the visiting staff. I did this myself in practice afterward, but found objections to the device in that it caused the insertion of one more ingredient in a formula which might already be complex enough, and in that often too much alkaline salt was given. Some five years ago I began directing patients to measure out their dose of bromides or of iodide into a glass, and add a liberal quantity of Vichy water, from one-half to a whole glassful. Gradually I adopted this as a vehicle in all cases, and can now testify to the excellent results of this practice from a three years' large experience. When the patient resides in a city or large town, I direct him to procure the artificial Vichy water in syphons, which is now so widely manufactured. Some of these imitation waters are very honestly made nearly like the known composition of the waters of Vichy, and others, the majority, I suppose, are carelessly compounded. At any rate, the

syphons contain a solution of bicarbonates of sodium and potassium highly charged with carbonic acid gas, and this is sufficient for our purpose. For patients living where the syphons cannot be procured, or for patients who travel much, I direct the purchase of the effervescent Vichy salts, either of American or foreign manufacture. A teaspoonful of the salts in a glassful of cold water makes a sparkling glass of Vichy water, in which the medicine can be mixed.

In the case of patients who cannot afford to buy these preparations, I recommend that a good-sized pinch of bicarbonate of sodium be added to a glass of water. The advantages which I claim for this method of giving bromides and iodides in weak alkaline waters surcharged with carbonic acid are two-fold :

First, the supposed irritating effects of the salts upon the gastric mucous membrane is reduced to a minimum if not absolutely neutralized. This statement is theoretical, but, practically, I am able to state that I almost never observe gastric or gastro-intestinal disorder while giving full or even very large doses of the salts to patients of various ages. I am thus enabled to administer from sixty to one hundred and more (5. to 10.) grains of the bromides in the day; and even when bromism occurs, the gastric symptoms are almost *nil.* The iodide of potassium I have thus given in doses varying from small doses to ℥i or 32. in the day, without indigestion. Occasionally for severe cerebral symptoms, I have caused children to have a dose of 5. three times a day, with only good results.

Second, the taste of the bromides and iodides is considerably masked by the sparkle and sub-acid taste of the effervescent drink. Many patients have thanked me warmly for having substituted a simple solution of bromides (or of iodide of potassium) given in Vichy water for the classical mixtures which they had formerly taken.

I should add that the salicylate of sodium is well taken in this way: a powder of the size required, 1. to 3., is dissolved in a glassful of Vichy water. This covers the disagreeable sweetish taste of the salt, and, I believe, favors its complete absorption.

In some simple cases of epilepsy I give only one dose of bromide of potassium in the day, at bedtime or on rising. In such cases I prescribe the medicine as a powder of from 2. to 4. or more, to be taken in a glass of Vichy water.

In some neurasthenic cases, and some cases of oxaluria with insomnia attended by restlessness, I have obtained excellent results from the use of a powder containing (usually) 2. each of salicylate of sodium and bromide of potassium taken in a glassful of Vichy water.

THE TENDON REFLEX IN GENERAL PARALYSIS OF THE INSANE.*

By J. C. SHAW, M.D.,

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AT a meeting of this Society held in June, 1879, I presented a short communication on the tendon reflex in the insane.[1]

Since that time I have made a more special study of this reflex as it is found in general paralysis of the insane, and it is my object now to present the results of my observations.

In 1879 I had only examined it in ten (10) cases of general paralysis. I then stated, and which was in accord with views previously expressed by Prof. Westphal, that whenever the tendon reflex was absent we were justified in deciding that sclerosis of the posterior columns existed, and I presented sections from the spinal cord, in one case, in confirmation of this. I also ventured the statement that a light cortical sclerosis would also abolish this reflex. My studies up to this time have confirmed those ideas.

Up to that time I had only seen the reflex, normal or absent, in these cases, but my subsequent studies, made upon a much larger number of cases, have brought under my observation the reflex in its normal condition, the reflex absent, present to a slight degree, and exaggerated; and it

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* Read before the American Neurological Association, at its seventh annual meeting in New York, June, 1881.

[1] ARCHIVES OF MEDICINE, vol. ii, p. 46.

is under these four or rather three modifications that we shall study it. I have studied the reflex in seventy-one (71) cases in men and eleven (11) cases in women, and have examined the spinal cords in eighteen (18) cases.

In the seventy-one cases of men we found it as follows:

Normal	in 28 cases
Slight	in 8 "
Absent	in 13 "
Exaggerated	in 22 "

In the eleven women it was as follows:

Normal	in 7 cases
Absent	in 1 case
Exaggerated	in 3 cases

Of the eighteen cases in which autopsies have been made and the spinal cords examined, I shall give histories in the briefest manner possible, and shall make no attempt whatever to give a description of the histologic changes in these cords at this present time, but speak of the lesions as they occupy regions of the cord, as my object is simply to connect, if possible, the alterations in this reflex with certain definite localized lesions; the absence of this reflex having been considered as a symptom almost pathognomonic of locomotor ataxia, and its exaggeration as a distinctive symptom of spastic spinal paralysis and lateral amyotrophic sclerosis.

All these spinal cords have been examined after hardening and mounting according to Lockart Clarke's method.

Case 1.—P. E., age 27, admitted August, 1875, attack having begun six months previously; he is excited, and has delusions of wealth and greatness; when I first saw him the disease was far advanced; he was feeble, ataxic in speech and gait, had marked tremor of facial muscles, left pupil slightly contracted, and delusions of wealth; he gradually became exhausted, and died.

Post-mortem showed decided lesions of the meninges and brain, also spinal cord, the part which alone will concern us for the present, and examination of which showed sclerosis of the posterior columns.

CASE 2.—H. D., admitted July 15, 1879, age 54. On admission, well-marked delirium of extravagance; says he owns all the world except Canada and San Francisco, which belong to his brother. Feb. 18, 1879, delirium of extravagance still continued, marked ataxia of lower extremities, difficulty in speaking, tremor, lancinating pains in legs; has hypochondriacal ideas. Dec. 6, 1879, has an epileptiform attack confined to left side; conjugate deviation of eyes to left side; tendon reflex entirely absent. April 12, 1880, has another epileptiform attack, confined to right side. Oct 23, 1880, to-day has epileptiform attack confined to right side; conjugate deviation of eyes to right side. Oct. 31, 1880, patient dies.

Examination of the spinal cord shows sclerosis of the posterior columns.

CASE 3.—G. F. S., age 41, admitted June 21, 1877. Is quite blind on admission; says he is blind, but could see if he wore blue glasses; he is the greatest musician in the world, etc. When seen by me he was in a state of advanced general paresis, and quite demented, filthy in habits; has never had an epileptiform attack (?); tendon reflex absent. Patient died August 10, 1879.

Examination of the spinal cord shows sclerosis of the posterior columns.

CASE 4.—R. O. B., age 32, admitted March 7, 1879. Pupils normal; has well-marked ataxia of gait; is demented; no delusions of extravagance; tendon reflex absent. Aug. 17, 1879, in the morning had epileptiform convulsions, both sides of body convulsed, lasting all day, with intermissions; he died the next day.

Examination of spinal cord shows sclerosis of the posterior columns.

CASE 5.—I. L., age 55, admitted June 21, 1879. Delirium of extravagance, well-marked ataxia of lower extremities, right pupil contracted, general tremor, advanced stage of paresis; in very feeble condition; no epileptiform attacks at any time; tendon reflex absent. Died July 23, 1879.

Examination of the spinal cord shows sclerosis of the posterior columns.

CASE 6.—H. P., age 34, admitted Feb. 4, 1878. Intemperate; duration of attack said to be one year; has been suicidal and homicidal; delirium of extravagance; when seen by me is in an extreme state of dementia; tendon reflex normal; has occasional epileptiform attacks. Died March 17, 1879.

Spinal cord found normal.

Case 7.—C. Van S., age 32, admitted April 1, 1879. Intemperate, pupils normal, in advanced stage of dementia paralytica, marked general tremor, tendon reflex normal. Died Sept. 20, 1879.

Examination of spinal cord shows no lesion.

Case 8.—T. G. C., age 33, admitted July 18, 1879. Intemperate, pupils normal, duration of attack said to be two years. Sister very nervous, and father intemperate. Delirium of extravagance, which passed off almost entirely after he had been in asylum a short time; all that remains of it is that he says he paid a woman fifteen weeks' board in advance; tremor of facial muscles and tongue; tendon reflex normal; slight difficulty of speech. Died of pneumonia Sept. 2, 1880.

Spinal cord shows no lesion.

Case 9.—F. M., age 32, admitted Oct. 26, 1879. Delirium of extravagance, tendon reflex normal, marked tremor, decided dementia, duration of attack said to be eight months. Epileptiform attacks. Died in one, March 18, 1880,

Spinal cord shows no lesion.

Case 10.—J. D., age 49, admitted April 10, 1878. Intemperate; this patient was seen by me at least eight months before his admittance to the asylum; has had injury to head a few years before from fall out of a wagon; marked delirium of extravagance; marked difficulty in speech. No epileptiform attacks, but frequent hemi-paretic attacks, which would almost completely pass off. Tendon reflex exaggerated; marked tremor. Sept. 16, 1879, had a paretic attack of left side; he became gradually weaker, and had to keep his bed; there followed difficulty of swallowing and respiration, light coma passing into stertor, and he died Sept. 20, 1879.

Examination of the spinal cord shows symmetrical degeneration of the lateral columns.

Case 11.—U. Van V., age 60, admitted March 15, 1879. Light delirium of extravagance, marked general tremor, frequent hemi-paretic attacks of one side and then the other, decided difficulty of speech. Died Oct. 31, 1879.

Spinal cord shows symmetrical degeneration of the lateral columns.

Case 12.—G. C., age 34, admitted Nov. 26, 1879. This patient was seen by me many months before his admission to the asylum; he is depressed and melancholic; no delirium of extravagance;

marked hesitancy in speaking ; subsequently delirium of extravagance ; has hemi-paretic attacks ; tendon reflex exaggerated ; never epileptiform attacks ; toward end of disease rigidity of muscles ; contracture. Died Feb. 14. 1881.

Spinal cord shows symmetrical degeneration of the lateral columns, with dilatation of central canal in cervical region.

CASE 13.—O. W. P., age 44, admitted Jan. 30, 1880. Intemperate, left pupil contracted, marked tremor, unsteady gait, very marked hesitancy in speaking. On admission, reflex is found normal; in April, 1880, reflex found exaggerated, and before death there appears some contracture. March 28, 1881, hemiparetic attack of left side quite complete. Died April 17, 1881.

Examination of the spinal cord shows symmetrical degeneration of the lateral columns.

FIG. 1.

Diffused central sclerosis. Patellar reflex exaggerated. Case 14.

CASE 14.—L. M., age 45, admitted Feb. 26, 1880. Advanced stage of dementia paralytica, marked hesitancy in speaking, marked tremor of tongue and facial muscles, a good deal of unsteadiness of gait, tendon reflexes exaggerated, extravagant ideas from time to time, hemi-paretic attacks, never had epileptiform attacks. Died Jan. 30, 1881.

Examination of spinal cord shows a diffuse sclerosis, shown in fig. 1.

CASE 15.—D. McC., age 46, admitted April 26, 1880. Left pupil contracted, marked tremor of tongue and facial muscles, decided hesitancy in speaking, marked defect of memory, advanced stage of general paresis, slight delirium of wealth and extravagance, tendon reflex exaggerated, and at last some rigidity and contractures. Died Jan. 11, 1881.

Examination of the spinal cord shows symmetrical descending degeneration of lateral columns.

CASE 16.—F. D., age 43, admitted May 31, 1880. Intemperate, excessive tremor of tongue, no pupillary changes, marked dementia on admission, hemi-paretic attacks, but no epileptiform attacks, tendon reflex exaggerated. Died February 11, 1881.

Examination of the spinal cord shows descending symmetrical degeneration of the lateral columns.

The condition which is found in these cords is illustrated roughly in fig. 2. The sclerosis is not strictly confined to the lateral columns, but is much greater there than anywhere else in the other regions of the cord.

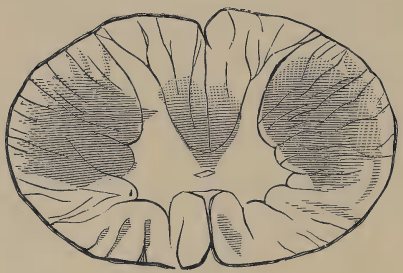

FIG. 2.

Symmetrical sclerosis of the postero-lateral columns and slight sclerosis of the deeper part of the posterior columns. Patellar reflex exaggerated. Cases 11–17.

CASE 17.—P. B., age 60, admitted October 16, 1880. Speech thick, partly aphasic; marked tremor; rigidity of muscles; quite demented; tendon reflex exaggerated; marked trophic changes before death. Died November 13, 1880.

An examination of the spinal cord in this case showed a well-defined symmetrical degeneration of lateral columns.

CASE 18.—W. H. C., admitted September 14, 1878. Marked delirium of extravagance; hypochondriacal ideas in advanced stage of the disease when seen by me; tendon reflex slight, and subsequently is entirely absent, contracture very marked for several months before death, which took place June 2, 1880.

FIG. 3.

Cortical sclerosis of the spinal cord with chronic meningitis. Patellar reflex lost. Case 18.

Examination of the cord shows a very deep cortical sclerosis with extensive chronic spinal meningitis.

It will be seen from these cases with *post-mortem* examination, that whenever the tendon reflex is abolished, we are quite safe in predicting that sclerosis of the posterior columns exists, and when the reflex is found to be very slight, that the posterior columns are the seat of disease in all probability, and that later, when the disease is more advanced, it will be entirely abolished.

In those cases in which the reflex is found to be normal,

post-mortem examination shows no lesion of the spinal cord.

In the cases in which the reflex is found exaggerated, we find symmetrical secondary degeneration of the lateral columns, or we find a diffuse myelitis which involves more or less of the white matter, and the lateral columns are always involved.

We may have cases in which the tendon reflex is slight and ultimately becomes entirely abolished, and having for its pathological basis a marked cortical sclerosis.

Moreover, we may even have the tendon reflex entirely abolished, and there be present marked contracture not depending in any manner upon a lateral sclerosis, but depending upon cortical sclerosis due to marked chronic spinal meningitis.

We have watched cases in which the reflex has become slighter and slighter, and at last disappeared.

In some of the cases in which the reflex was found exaggerated, and the cord subsequently examined microscopically, it was found that there was a sclerosis of light character almost everywhere, but most marked in the lateral columns, and the posterior columns near the posterior commissure also had a light sclerosis. I have from this been led to conclude that to abolish the reflex, the sclerosis of the posterior columns must be quite extensive, or there must be a marked cortical sclerosis, and the reflex thus find its point of obstruction in the posterior roots.

The exaggerated reflex is closely connected with two prominent symptoms in this disease. Those cases in which there are marked difficulties in speech, hesitancy, stuttering up to complete inability to speak (not aphasia proper), are the cases in which is always found, sooner or later, exaggerated tendon reflex.

And it is in those patients who have the marked difficul

ties in speech and the exaggerated tendon reflex that we find almost invariably hemi-paretic attacks, and comparatively rarely epileptiform attacks. There is, therefore, a direct connection between these difficulties in speech, the hemi-paretic attacks, and the exaggerated tendon reflex, and this is susceptible of pathological demonstration, and will be the subject of a communication from me at a future time.

A CASE OF ASYNCHRONOUS CONTRACTION OF THE CARDIAC VENTRICLES, WITH REMARKS UPON REDUPLICATION OF HEART SOUNDS.

By FREDERICK P. HENRY, M.D.,

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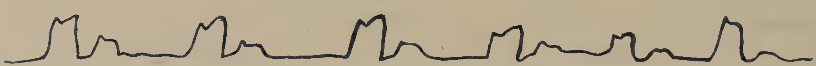
THE publication of isolated cases of disease is alone justified by one of two reasons: first, as affording undeniable facts in support of some physiological or pathological doctrine; and, secondly, on account of their extreme rarity. I can, I trust, successfully advance both of these reasons for the publication of the following case. As to its rarity, I need only remark that in a rather extended search among text-books and periodicals, I have found but one other similar case recorded. It is contained in *Virchow's Archiv* for 1868, and bears the following title: "Ungleichzeitige Contraction beider Ventriklen." Mitgetheilt von Prof. E. Leyden in Königsberg.

My case was a multipara, æt. 34, who was a patient in the Episcopal Hospital when I took charge of the medical wards on April 1, 1879. She had had eight attacks of articular rheumatism, the first when twelve years of age, and the last, three years previous to admission, at which time the heart trouble began. There was no œdema; respiration, in the recumbent position, was tranquil, and the urine was free from albumen. On examining the heart, I detected a distinct mitral regurgitant murmur, and also what at first appeared to be a very irregular action of the cardiac

muscle. As the pulse was only 48 per minute and perfectly regular, I was led to study the action of the heart more minutely, and very soon discovered that the apparent irregularity was due to a separate action of the ventricles. The pulse was, as has been said, 48 per minute, and over the heart's apex could be counted 96 distinct pulsations, succeeding each other at regular intervals and each apparently composed of a complete cardiac revolution. The cardiac sounds were four in number, the first accompanied by a murmur loudest at the apex, and their rhythm was irregular, the first two and last two succeeding each other more rapidly than did the second and third ; that is to say, there was a distinct pause between the separate action of the ventricles, but decidedly



No. 1.—Tracing of apex beat of heart.

No. 2.—Pulse tracing of right brachial artery.

shorter than the regular pause occurring at the close of the complete revolution. The intensity of these sounds also varied in degree ; the first and third, however, namely, those due respectively to the closure of the mitral valve and the contraction of the left ventricle, and to the closure of the tricuspid valve and the contraction of the right ventricle, being nearly equal in intensity.

The accompanying sphygmographic tracings, kindly made by my colleague, Dr. Louis Starr, explain the condition far better than can any mere verbal description.

The patient left the hospital shortly after I saw her for the first time, but returned a few weeks later with all her symptoms

changed for the worse. She began to get worse about two weeks after leaving the hospital. On admission for the second time, on May 19th, she complained of severe præcordial distress, which had then lasted for five days. She also suffered from marked dyspnœa. There was also a considerable degree of œdema of the lower extremities, together with ascites, the abdomen around the line of the umbilicus measuring 33 inches. It was now observed that the asynchronous action of the cardiac ventricles was not a constant symptom, but came and went without any apparent cause. I extract the following from notes taken at the time by the then resident physician, Dr. H. H. Bickford :

May 28th. Pulse 44, with double apex beat of heart.

June 6th. Heart and pulse are synchronous.

June 7th. Double cardiac beat to one beat of pulse.

June 14th. Œdema in feet and legs all subsided.

Measures 30 inches around abdomen. Double apex beat.

June 17th. Pulse 48. No præcordial pain. Pulse and apex beat synchronous. Appetite good. Bowels moved once daily.

June 19th. Discharged at her own request.

There are three principal means by which reduplication of cardiac sounds may be produced. The first, most common, and best understood, is the asynchronous closure of the aortic and pulmonary valves, and is not a very rare phenomenon. It occurs both physiologically, as has been shown by Potain, and in disease when, from any cause, the normal ratio of aortic and pulmonary tension is destroyed. This asynchronous closure of the semilunar valves gives rise to the *bruit de rappel* of Bouillaud, and inasmuch as it is composed of a long sound, followed by two short ones, it has been called a dactylic sound, and may be represented by the usual symbol for the dactyl, — ◡ ◡.

The second principal cause of reduplication of cardiac sounds is due to an abnormal action of the left ventricle, giving rise to the *bruit de galop*, also first recognized and named by Bouillaud, but afterward more minutely studied and described by Potain. From its resemblance to the foot of Greek and Latin metre known as the anapest, it has been

spoken of as an anapestic sound, and may be represented with considerable accuracy by the usual symbol for the anapest, ◡ ◡ —.

The extra heart sound which gives rise to the *bruit de galop* is presystolic, and it has been demonstrated as a movement by Potain, by means of the cardiograph, and the same observer has shown that this movement is the distension of the ventricle, accomplished and completed by the contraction of the auricle.[1] Potain shows in confirmation of this view that this presystolic movement coincides with the jugular venous pulse constantly encountered in these cases. Potain's explanation of the mode of causation of the *bruit de galop* may be summed up as follows: The exaggerated arterial tension that exists in cases of interstitial nephritis causes diminished venous tension. Owing to this the ventricles are more incompletely filled during the first period of the diastole, the presystolic period, than usual. That is to say, there is more work than usual thrown upon the auricle. This causes a sudden distension of the ventricle, and also the exaggeration of the jugular pulsation constantly encountered in these cases.

Sibson, who has encountered this anomaly, regards it as due to an asynchronous closure of the auriculo-ventricular valves, but Potain, without denying the occurrence of asynchronous closure of these valves in other conditions, contends that the special abnormality known as the *bruit de galop*, is otherwise produced, for the three following reasons:

1. The abnormal sound has not the timbre of valvular closure.

2. It does not predominate, as it should in that case, in the region of the right cavities.

3. Potain declares that he has heard, in the same cardiac

[1] *Union Médicale*, 1876, 3ème série, t. 21, p. 324.

revolution, the asynchronous closure of the auriculo-ventricular valves, and also the *bruit de galop*.

Of the three reasons above given, the second is, to my mind, the most convincing, but it seems to me strange that so acute an observer should have omitted to refer to the double impulse, which when felt or demonstrated with the sphygmograph, is almost pathognomonic of these cases.

On reading the interesting papers of Potain, the impression is derived that the *bruit de galop* is alone encountered in cases of interstitial nephritis, but that this is a mistake, is proved by the following history of a case recently under my care at the Episcopal Hospital.

Louis P., æt. 30, a tailor, of intemperate habits, was admitted April 20th. His feet, legs, thighs, and scrotum were highly œdematous, and there was a slight degree of ascites. The face also was the seat of slight œdema. The heart was enlarged, the apex beating in the fifth space, one inch to the left of the nipple line, and, on auscultation, the *bruit de galop* was heard in perfection. The urine was loaded with albumen. The man died on April 24th, and I had the opportunity of making an autopsy.

The kidneys were flabby and weighed respectively 6½ and 7½ ounces. Their capsules were readily detached, leaving a perfectly smooth surface, mottled with large patches, varying from a pale-pink to a yellowish-white. Under the microscope they were found to present a perfect picture of extreme chronic parenchymatous nephritis. There was no healthy epithelium to be seen. The tubes were blocked with granular débris, and where the section was thinnest, the picture presented by the outlines of the tubules denuded of epithelium, closely resembled that of a brushed-out section of lymphatic gland. Finally, there was no increase whatever of the interstitial connective tissue.

The heart was of flabby consistence and weighed fourteen ounces, the enlargement being mainly confined to the left ventricle, which was decidedly dilated. Examination with the microscope showed the fibres to be in an advanced stage of granular degeneration. In only a few of them could faint traces of striation be detected. The valves were all competent and healthy.[1]

[1] From notes taken by the resident, Dr. R. P. Robins.

Of two other cases under my care, in which the *bruit de galop* existed in typical form, in one there is no doubt as to the kidney disease being acute parenchymatous nephritis. This was evident from the short clinical history, the extreme anasarca present, the occurrence of convulsions, the high degree of albuminuria, and the presence in the urine of great quantities of epithelial and highly granular casts. This patient is at present, June 3d, an inmate of the Episcopal Hospital.

In the other case alluded to, the enormous œdema of lower limbs and scrotum, and the large amount of albumen in the urine were sufficient to exclude the diagnosis of interstitial nephritis.

Although I accept Potain's explanation of the mechanism of the production of the *bruit de galop*, I am compelled, from the study of the cases above referred to, to reject his statement of its association solely with interstitial nephritis.[1] The *bruit de galop* may exist whenever there is excessive arterial tension with diminished venous tension, which condition is most frequently present in diseases of the kidney. It is my opinion that this sound is often overlooked, and that the heart's action is described as "irregular," when it is not so in the strict sense of the term. Attention should always be paid to the relative action of the heart and pulse. If the heart's action is apparently irregular, while there is no corresponding irregularity of the pulse, the case should be carefully investigated, and if this be done, the least expert in the diagnosis of cardiac disease may occasionally detect one or other of the interesting anomalies described in this paper.

A few words as to the diagnostic significance of the *bruit de galop*.

[1] "On trouve au cœur, chez les malades atteints de néphrite interstitielle, un bruit spécial qui est le bruit désigné par M. le professeur Bouillaud sous le nom de bruit de galop."—*Union Médicale*, 1876.

Potain considers this abnormal sound to be so high in the scale of diagnostic importance that occasionally it may call attention to the existence of an interstitial nephritis that might otherwise continue unsuspected. This is certainly according it a high rank. I can readily conceive of the following series of circumstances in the examination of a patient; in fact it has been my own experience, and it is perhaps to something similar that Potain alludes. For example, in proceeding systematically with the examination of a patient, the lungs and heart are usually the first organs to which attention is directed. In so doing, the *bruit de galop* may be detected, and later, on testing the urine, albumen, or casts will be found. It is hardly fair to say in such a case that the *bruit de galop* has been of great diagnostic importance. It would be, if the albumen and casts were absent, and yet a latent disease of the kidney could be certainly determined from the existence of this sound alone. For my part, I regard it as of about as much diagnostic value as the retinitis nephritica, of which oculists were wont to talk so much in the first flush of their discovery, and of which so little is said to-day. I have yet to see the first case of Bright's disease that can be diagnosticated *solely* by the ophthalmoscope.

I have entered so fully into the preceding details in order that experts in heart disease may not hastily conclude that the case I report is one of the *bruit de galop*. A comparison of the sphygmographic tracings with those in Potain's papers will show the wide difference between them. In my case also there was no kidney disease that could be detected by the most careful application of the ordinary tests.

I would suggest that the sound produced by the asynchronous contraction of the ventricles be spoken of as a double iambic sound, and that it be represented by the

symbol for the double iambus, — ᴗ — ᴗ, and in so doing, I refer to the sounds independently of any murmur that may be associated with them. In the only two cases with which I am acquainted, Prof. Leyden's and my own, there was valvular disease, of rheumatic origin, giving rise to one or more murmurs. In Leyden's case the valvular disease was more than usually complicated. "There is," says he, "certainly a stenosis of the aortic ostium and a tricuspid insufficiency, probably also a stenosis of the ostium tricuspidale."

There is one form of valvular disease which, I think, entirely prevents the formation of the *bruit de galop*, namely, mitral obstruction. The first sound of the *bruit de galop* is due to a diastolic pulsation of the ventricle, caused by an abnormally energetic contraction of the auricle in the presystolic period, and in order that the auricular contraction may have full effect, the mitral orifice must be unobstructed. I do not say this unadvisedly, for in a case of mitral obstruction that was under my care about two years ago, the left auricle had become so hypertrophied that its pulsation could be distinctly felt, and aneurism was at first suspected. This was readily excluded, and mitral obstruction immediately diagnosticated. Notwithstanding this abnormally powerful action of the left auricle, there was never detected the slightest diastolic pulsation of the left ventricle.

It had been my purpose to refer to some interesting points concerning the action of the healthy heart, suggested by this case of asynchronous ventricular action, but this article has already grown beyond its intended limits.

EDITORIAL DEPARTMENT.

HIGHER MEDICAL EDUCATION IN NEW YORK.

III.

THE SYSTEM OF CLINICAL TEACHING IN COLLEGES.

I include clinical teaching in medical schools under the general head of higher medical education for two reasons :

First, for the general reason that clinical study logically follows the elementary medical studies, and is continued, after graduation, *ad infinitum.*

Second, because I am quite sure that a number of clinics in every college are attended by practitioners for the purpose of learning things that are new, or things which have practical importance.

It is my purpose to show (1) that the vast material at the disposal of clinical instructors in New York is not utilized in such a way as to afford the advanced medical student systematic instruction in the different departments of medicine, and (2) that, so far as I know, no attempt is made to coördinate the clinical and didactic lectures during the course of study.

A college clinic is usually organized as follows : a clinical professor or lecturer has charge of the clinic, assisted by two or more younger physicians. The attendance of patients varies according to the punctuality of the physicians and the care and considerate treatment they bestow upon patients,—usually there is an abundance of "material," as we call it. As a rule, no case-books are

kept, and the large majority of patients are seen only by the clinical assistants. A few minutes before the time appointed for the lecture the professor asks his assistants for "interesting" cases, or sometimes selects them himself. In the lecture-room a series of three, four, or five such cases is shown to the class without classification. In a medical clinic, for example, cases of rheumatism, paralysis, phthisis, etc., may be considered in one hour's time. Occasionally, for some lectures requiring subjects to submit themselves to painful or annoying demonstrations before the class, patients are sought beforehand. Inevitably, during a session, the student sees a most tiresome repetition of cases under such an unsystematic plan; cases of dyspepsia or rheumatism may be paraded *ad nauseam* before the class.

There are other serious defects in our college clinics. Nearly always the professor lectures upon an unstudied case, and is obliged to pass over a number of important data necessary for accurate differential diagnosis. For example, a case of headache is talked about before the class without the necessary examinations of the urine, of the state of optic refraction, etc.,—elements which are often indispensable to a correct judgment. In many cases there are delicate questions to be asked about sexual symptoms, syphilis, etc., which many patients will never answer truthfully in public. Often, too, dealing with almost unknown cases, the teacher spends a quarter of an hour or more in extracting a tangled history of symptoms from a patient, and then realizes that the case is unimportant, or at any rate pointless for clinical purposes.

Very often the clinical remarks made are mere remarks, a desultory talk about the cases, others like it, their treatment, etc., showing on the professor's part a total want of appreciation of the functions of a college clinic.

Another evil of our present plan is that cases are seldom shown a second and third time after a first study in public. This is often unavoidable, as clinic patients are provokingly uncertain in their attendance. Still, by care and by the aid of clinical assistants or of medical students, the cases can be hunted up and induced to

come again to enable the class to observe the progress of a disease or the action of remedies.

It has long seemed to me that however inferior college clinics must be to hospital clinics held over bed-ridden patients, much more instruction might be extracted from them than is now done.

This improved teaching might be attained by applying the following propositions to clinical work :

I. Recognizing that the principal function of an " out-door " clinic, or college clinic, is to afford students an opportunity of studying methods of examination and the diagnosis of diseases.

Considerations of pathology and of therapeutics, except, perhaps, in surgical and special clinics, should be relegated to the background, and made prominent only in cases of simplicity, or cases which are likely to return to the clinic.

Under the head of methods of examination, I would include teaching the art of questioning a patient so as to obtain the data for a history of his case and for a diagnosis. This embraces a peculiar kind of logic, a train of silent reasoning which the expert examiner is carrying on all the time while talking with the patient, and which enables him, by the aid of past experience, to follow up useful clues and take up at the proper moment hints which the patient may, perhaps unconsciously, have dropped in his replies. In many cases a conversation of ten minutes enables the professor to seize the capital symptoms and the etiological factors of a case, and to write them upon the blackboard for further use in discussion. This logic of examination varies in each department of medicine, being in some cases superior to the physical examination, while in others it is subordinate.

It is also desirable that the clinical teacher should briefly describe all the instruments which he uses in examining organs and testing functions, and give repeated demonstrations of their use.

A most important, perhaps the most important, subject of study at such a clinic is what I may call analytical semeiology. By this I mean the accurate definition and close analysis of symptoms. How often do we hear physicians of experience speak of symp-

toms in such a way as to show that they do not really understand those signs, those characters through which a disease is classified; for example, what confusion about the terms numbness, ataxia, hallucination, etc. Besides an accurate definition of a symptom, and its demonstration when possible, the teacher should explain to the student the anatomical basis of the symptom, and the physiological function of which the symptom is (often) the perverted expression. This opens a wide and legitimate field for giving students repeated lessons in those portions of anatomy and physiology which the practical physician must know at his fingers' ends. Such a study of the anatomical and physiological basis of symptoms also opens the way in several departments of medicine (diseases of the thoracic organs, of the nervous system, etc.) to accurate regional diagnosis, or diagnosis of localization of disease.

Next in order of exposition comes the mode of grouping, or association of symptoms. This should be taught both positively and negatively, and in so doing there will be ample opportunity to show how delusive and misleading is the so-called "pathognomonic symptom." By the positive mode of studying the association of symptoms, I mean showing how symptoms and so-called physical signs obey certain tendencies of association and form a "symptom-group," which though not the disease itself, yet often serve for its classification and demonstration. By the negative study of symptoms in their relations with other symptoms, I mean showing how one symptom may be a part of several disease symptom-groups, and may even be caused by fundamentally different pathological conditions.

An improvement which I would suggest in college clinical teaching, and it seems to me of considerable importance, is the much greater use of the blackboard. Now, in most clinics, the blackboard is only used for normal and pathological sketches or diagrams. What I think should be generally done is, with the aid of several blackboards, to write down (1) a summary of the history of the case, (2) a summary of the chief symptoms as observed in the patient, (3) the necessary anatomical diagrams or

sketches, and sometimes (4) an important law or definition. With these data before them in writing, a class of students can intelligently follow the remarks which the professor makes, can carry out in concert with him the logical processes of assimilation and differentiation by which the diagnosis is reached. Without such objective reproduction of a case upon the blackboard, I firmly believe that, for all but a very few unusually well-trained minds in the audience, the clinical teaching is foggy and unprofitable. The class may "see" an endless series of cases in a session, but would not the "understanding" of fewer selected cases do much more toward their training for practical life? It may be said that all this writing on the blackboard is an useless drudgery, that the student should remember the points of a case. This is all very well for the simplest cases, presenting only a few physical characters for study, but when we come to deal with serious medical and surgical cases, in which enter a great number of considerations, where a diagnosis is only to be reached by induction from many data and by close inferential reasoning, or if we are studying cases on the borderland of new knowledge, I say that trusting to the memory of a mixed class of students is altogether vain,—it is overrating their mental powers, and by paying them this empty compliment we deprive them of what they come to us to obtain, viz.: training. Not only is it true that students seldom show ability to retain the data of a complicated case, but it is also true of medical men. How often do we see in the course of discussion at medical societies, members of fair standing ask questions and make remarks which conclusively prove that they have not *understood* the case presented or the paper read a few moments before they rose to speak. Perhaps I am not exaggerating if I say that the ability to comprehend and retain the elements of an oral medical communication is an evidence of unusual mental power and of careful training. How can we presume these attributes to exist in our pupils? No; I maintain that the young men who attend our clinics should have every thing presented in the most objective and tangible manner possible, should be made to participate in our

diagnostic reasoning, and should be given every opportunity for note-taking.

The practice of taking notes at clinics is, it seems to me, very important, and it is not open to the same objection as note-taking at didactic lectures. In a clinic conducted on the plan which I suggest, there is much beneficial repetition, time is consumed by writing on the blackboard, so that the student is not hurried in noting. The record of a number of cases thus analytically studied must prove invaluable to the intelligent and earnest student. At any time he can turn to such a case-book, and by its guidance conduct a course of reading—reading about the symptoms themselves, reading on the anatomical and physiological points noted down, reading on the pathology and pathological anatomy of the cases, etc.

I would ask every candid reader to compare the possible results of such clinical work with that following the exhibition of cases, with "remarks," as practised now at college clinics.

II. The college clinics should be made to supplement the great didactic chairs of the school. In other words, clinical and didactic teaching should be carefully correlated.

At the present time clinical teaching in our medical schools may, with perhaps some exceptions, be characterized as haphazard. Whatever turns up in the way of "interesting cases," is shown to the class of students. No attempt is made to follow a system in the presentation of cases, or to illustrate in the clinics the subjects which are, at the time, being taught didactically. Yet with foresight and a little trouble all this might be remedied. At the beginning of the session a conference of the didactic and clinical teachers in a school should be held, and a programme of didactic lectures upon medicine, surgery, and a few special subjects, constructed. If any changes become necessary in the order of lectures, the assignment of subjects from week to week, a memorandum should be sent to the clinical teachers interested. With such a coöperation as to plan, by some exertion, perhaps occasionally at a small expense, the clinical teachers could provide cases in illustration of the didactic lectures at the proper

time, *i. e.*, immediately after these have been delivered. Let us, by way of illustration, suppose that in the second week of January the professor of medicine has lectured upon organic diseases of the heart. During the third week of the same month, the professor of clinical medicine in the college could, by making an effort at collecting patients beforehand (even if necessary sending carriages for some of them), exhibit to the class a number of cases typical of the chief organic cardiac diseases—of all those which allowed the patients to leave their homes.

But, further, the clinical teaching outside of the college might thus be coördinated, to the immense benefit of the class. The professors of clinical medicine, physicians in the various hospitals of the city, should likewise be notified of the subject under study that second week in January, and they could select and arrange the material for hospital clinics upon organic cardiac diseases, thus enabling the students to see the bed-ridden, extreme cases of this class. If the services of outside clinical teachers could thus be coördinated and utilized, a medical school should have many attached to it, certainly at least one in each hospital. The title of professor of clinical medicine or surgery is one which most prominent hospital physicians would be pleased to have from a well-ordered medical school, and the conferring of the title, with perhaps a nominal honorarium, would be a small price for the school to pay for their services.

The clinical teaching of specialties would have to be independently arranged, yet even here the course could be systematized. The clinical professors of diseases of the eye and ear, of dermatology, or gynecology, of diseases of the throat, of diseases of children, etc., must in such a scheme be a law unto themselves. Yet even they should be kept informed of the weekly progress of teaching in the great didactic chairs, and often they would be able to illustrate the didactic lectures. For example, when the professor of medicine was lecturing on tuberculosis, could not the special clinics for diseases of the throat and for diseases of children place before the eyes of the students instructive examples of local and general tuberculosis? Otherwise, each special clinical

professor could plan his own course, classifying the cases which come within his specialty, and offering them to the class in a certain order, either one of his own devising, or one already known to the students as laid down in a text-book. In this way, it seems to me, that the student would learn more though he might "see" fewer cases.

I have followed such a plan, in the absence of any understanding between the didactic and clinical chairs in the medical school with which I am connected, for several years—in fact, since I began the clinical teaching of nervous diseases. I know the advantages of such a plan, and I also think I realize its drawbacks. Its advantages have been set forth in the preceding remarks. The objections to the plan of systematic clinical teaching in specialties are numerous but not serious. There is considerable difficulty in procuring cases, in engaging their attendance at a given clinic. One is sometimes disappointed, and that, too, after a solemn promise. Of course, if the patients who were expected to illustrate a certain lecture fail to put in an appearance, this lecture must be postponed, and cases out of order, rare or not, must be presented; or the opportunity may be taken to give a half didactic lecture on methods of examination, on previous cases, etc. Such breaks in the plan do not, in my experience, occur often enough to be serious. A second objection is that the lecture thus planned, and with its analytical study of cases, is less "interesting" or brilliant. I am ready to grant this, because I fully understand how the word "interesting" is employed by some students ; it is synonymous with curious, showy, or exciting. The method which I have suggested, obliges the lecturer to adopt a conversational tone, to repeat statements, to be exact in the use of words, to pause to give demonstrations ; all of which is opposed to oratorical display. It may also be urged that according to this plan the teacher has reached a diagnosis in the cases exhibited before they are presented to the class, and that the class is deprived of the privilege of seeing him make a diagnosis. This would be a valid objection if the clinic were for the purpose of "showing off" the professor's diagnostic skill, but for those who believe, as I do, that a

clinic is for the purpose of helping to train medical students, the making of a brilliant off-hand diagnosis by the teacher is vastly less important than a scientific analysis of a case, however " slow " it may appear to some members of the class.

III. College clinics might, it seems to me, also be used for the purpose of the personal training of individual students. This is, I believe, done to a certain extent, but it ought to be done much more. Earnest students can be invited to come to the clinic before and after the lecture, for the purpose of examining patients for themselves, under the guidance of one of the clinical assistants. In my experience assistants are always willing to take on this extra duty. The greatest difficulty in the way of any considerable extension of this personal instruction lies in a deplorably prevalent inertness of medical students. They are willing to crowd about an assistant who is examining a case, and "pick up" some knowledge easily, but very few are willing, in my experience, to do the only thing which can make such attendance profitable, viz., sit down with a patient, take his history in writing, mark the important symptoms, attempt a diagnosis, and submit the paper to the professor, or to one of the assistants, for correction and suggestion. The case thus worked up and corrected should be written at length, with diagnosis if necessary, and presented at the next clinic to the teacher. It is a matter of regret that so few, so very few, students seem to understand that three or four cases studied in this manner each week, would be worth more to them than the " seeing " of any number of cases in the usual way.

E. C. SEGUIN.

NEW BOOKS AND INSTRUMENTS.

Antagonism between Medicines and between Remedies and Diseases. Cartwright lectures for year 1880. By ROBERTS BARTHOLOW, M.D., Professor of Materia Medica and General Therapeutics in the Jefferson Medical College of Philadelphia, etc., etc. D. Appleton & Co. 1881. pp. 122.

The Cartwright lectures have been inaugurated most auspiciously by Dr. Bartholow. He has compressed into a narrow space a brilliant summary of the facts at present known in regard to one of the most fascinating questions of modern medicine. The demonstration of a precise antagonism between the action of drugs, has a double bearing on the theory of therapeutics. On the one hand, new practical resources are placed at our disposition, not merely to meet the accidents of poisoning, but, as we may hope, to combat symptoms similar to such accidents, when they have arisen spontaneously in the course of disease. But a further and more purely philosophical interest attaches to the study of the toxic symptoms, for the reason that their exact (remote) cause is known, and known to be an agent within our grasp.

The very existence of such a definite train of symptoms proves that we are able by external agencies to modify, in a given direction, the processes of a living organism. This fact is in formal opposition to the fundamental doctrine of medical Nihilism, which says : "It is absurd to attempt to modify anatomical conditions by means of drugs." In view of the palpable contradictions to this doctrine which the facts of toxicology affords, one of two conclusions must be admitted. Either the symptoms induced by poisons are independent of anatomical conditions ; or else by the administration of a drug, we *are* able to change the anatomical conditions of health to those characteristic of an artificial

disease. It is true that the conditions thus voluntarily induced are only similar to those of natural disease, and by no means identical with them. "We can," observes one of the most eminent authorities on artificial pathology, "imitate symptoms but not diseases. We can render an animal diabetic or epileptic, but we cannot create diabetes or epilepsy."[1] Nevertheless, this imitation is already of the greatest importance. And when, in studying the effects of one poison we find that they can be combatted by the appropriate use of another, and that this second poison can be shown to be capable of initiating a train of symptoms exactly the opposite in appearance to those which have been caused by the first, a horizon certainly opens before us of a rational therapeutics, destined to encroach more and more on the therapeutics of pure empiricism.

The hope of such a future is distinctly communicated by Dr. Bartholow, even in the title of his lectures. Consideration of the "antagonism between medicines" is immediately followed by discussions on an analogous antagonism "between remedies and diseases," and to this latter subject are devoted two out of the six lectures of the course.

It is on the "scientific application of the principle of antagonism to medical practice" that the author seems to rely, to reverse the severe judgment pronounced on materia medica by Bichat, in 1818. "It is a collection of incoherent opinions,—is, perhaps, of all the physiological sciences, that which most exhibits the contradictions of the human mind. In fact, it is not a science for a trained intellect; it is a shapeless mass of inexact ideas, of observations often puerile, of imaginary remedies strangely conceived and fantastically arranged. It is said that the practice of medicine is repulsive. I go further than this: it is, in respect to its principles taken from our materia medicas, impracticable for a sensible man." (Quoted, p. 13.)

Piquant indeed is the contrast between the uncertainty thus pungently described, and the exquisite precisions which, according to our author, may even now be predicted of so many therapeutical manœuvres. We would not deny Prof. Bartholow's energetic optimism. Optimism, even when exaggerated, often serves, like the flag of the color sergeant, to lead a substantial advance. But in estimating the resources at our disposal for the removal of disease, we think it is of great practical importance to bear in mind the (often unknown) *tertium quid*, which distinguishes

[1] Vulpian. Leçons sur les maladies de la moelle épinière.

morbid processes of spontaneous, *i. e.*, internal origin, from those which have originated in external influences, whether traumatic or toxic. The problem for somatic diseases is the same as for insanity : health failure at any one point of the organism very often, if not always, implies deviation of the entire organism from the norm. Hence, we are inclined to believe at least one cause of the frequent failure to allay spontaneous symptoms by remedies which have been successfully antagonistic to the same symptoms when artificially induced.

Did we follow Dr. Bartholow literally, we might infer that the different success in the two cases really depended on an absence of anatomical lesion as a basis for toxic symptoms. We are told to select our therapeutical agents on the basis of "physiological antagonism." And this "means simply a balance of opposed actions on the same tissue. It does not induce a change of structure. The opposing agents counterbalancing each other, the functional disturbance subsides, and the normal equilibrium is restored." (p. 11.)

But physiological actions are inconceivable except as the concomitant of molecular changes in the elements in function. The difference between each molecular change and gross palpable lesions of structure, is one of degree not of kind. An agent that causes arterial tension by relaxing the peripheric arterioles, determines a rearrangement of the molecules in their muscular coat. An antagonistic drug which should raise the tension by really acting on the same arterioles, must necessarily reverse the molecular arrangement effected by the first. The objective of the second drug is not the "opposing action of the first," but the tissue which has been modified by that.

But there are further objections to Dr. Bartholow's formula. We think it can be shown, even from his own summary of facts, that "opposed actions on the same tissue" never take place except in one direction. When a tissue or organ is paralyzed by any poison, it fails to respond to other poisons which ordinarily have a tendency to stimulate it. This failure is observed whether the paralyzing agent be administered first, or when the stimulating agent is in full operation. In the latter case, the stimulating poison is effectually antagonized. It is on this account that, as Dr. Bartholow himself remarks, the list of antagonisms effected by atropine is so large : it paralyzes so many "end-organs." Paralyzing the ciliary branches of the third nerve to the pupillary sphincter and to the ciliary muscle, atropine antagonizes all

drugs which cause myosis, either by stimulating the third nerve, or by antagonizing the ciliary muscle or circular fibres of the iris.[1] Thus, it antagonizes pilocarpine, eserine, muscarine, and the initial action of morphine. In the later stages of morphine poisoning, where vaso-motor paralysis of the iridian blood-vessels increases the myosis by turgescence of the iris, the counteracting effect is aided by its influence on the circulation. Now, in all the above cases, the antagonism of atropine to the myotic drugs is not reversed. When the pupil has been dilated by atropine, it is admittedly difficult to counteract it by any antagonist. In the most famous and thoroughly discussed antagonism, that between morphine and atropine, Dr. Bartholow declares that the pupil offers no sure guide, and that the action of atropine preponderates. Muscarine will not contract the pupil dilated by atropine. (See p. 63 of Lectures.)

According to Bartholow the "atropinized pupil resists the action of eserine" (p. 54). If, however, as Galezowski declares, eserine discs will contract a pupil so dilated, it would be by directly tetanizing the circular fibres of the iris; thus there would be no "opposed action" on the third nerve.

Quite similar observations hold true of the heart. Here again the "antagonism" of atropine is extensive and conspicuous, because it paralyzes the terminal fibres of the vagus in the cardio-inhibitory ganglion. Thus it antagonizes, in Dr. Bartholow's sense, by "opposed action on the same tissue," all the drugs which slacken the pulse by stimulating either the central or peripheral portion of the inhibitory apparatus. Thus, it is antagonistic to digitalis, to morphine in its early stages, to muscarine. But the experiment is classical in toxic experimentation, wherein the heart, arrested by muscarine, may be set to beating by atropine, while the atropinized heart altogether refuses to respond to muscarine. When morphine succeeds in reducing the pulse accelerated by atropine (and this is admittedly difficult), it does so by diminishing the excitability of the excito-motor ganglia. Here again, therefore, there is not "an opposed action on the same tissue," but a similar, *i. e.*, paralyzing action on a very different tissue.

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[1] Dr. Bartholow admits, in several places, that atropine "stimulates the radiating fibres of the iris;" but of this we know of no proof. The experiments upon the excised eye, we believe first performed by Brown-Séquard, only demonstrate that atropine acts on nerve terminations, and that the central communication of the third nerve is not essential. This is precisely analogous to its action on the terminal branches of the vagus, after section of the trunk.

Similarly, atropine will arrest the salivation caused by physostigma or pilocarpine, for it paralyzes the chorda tympani. When this paralysis has once been effected, salivation is no longer possible. Chloral will moderate the convulsions caused by strychnine ; there is no proof that strychnine will avert the respiratory paralysis threatened by toxic doses of chloral.

Dr. Bartholow admits this last fact with great surprise. We consider it rather as an illustration of a general law that we have already indicated, and which may be thus formulated :

"The response of an organ to a physiological or toxic stimulus may be prevented by paralyzing the organ. But paralysis of an organ cannot be antagonized by stimuli addressed to the organ, since the paralysis implies that susceptibility to impressions has been lost. Cure of paralysis can only be obtained by elimination of the paralyzing effect. During the process of elimination, the effects of the paralysis may often be combatted by stimulation of other organs remaining able to respond. This constitutes a net antagonism to the effects of the poison, often effectual, but always indirect."

It is this form of antagonism which is to be inferred from the "physiological basis" described by Dr. Bartholow. Part of this basis is afforded by the mechanisms which exist throughout the body for systemic alternation of functions, with consequent "restraint of activities within proper limits."

"If there were not some antagonism to the spasm centre, every trifling peripheral irritation would produce most extravagant reflex effects. * * * The movements of the vessels are regulated by a vaso-motor centre in the medulla. By the opposed action of the dilator and constrictor forces, the vascular tonus is maintained at the normal. A similar mechanism controls the cardiac movements ; there is a motor apparatus for carrying on the action of the heart, and a regulator apparatus for restraining the movements within proper limits. * * * If the arterioles suddenly dilate, the blood-pressure as quickly falls, but danger to the circulation is prevented by an increased action of the heart. * * * Here opposing forces maintain their equilibrium" (p. 21).

The presumption is that artificial antagonism to a given process in an organ will be best effected by acting upon the apparatus which provides for physiological antagonism to the same process.

If we apply this principle to some of Dr. Bartholow's favorite illustrations of antagonism, we shall discover quite a different interpretation of them from that given in these lectures. For in-

stance, atropine is said to "stimulate respiration," because accelerated respiration is a phenomenon induced by atropine. Hence atropine is considered a valuable antagonist to any poison threatening death by "respiratory paralysis."

Now, it must be observed, in the first place, that each of these opposed terms is not simple, but extremely complex. The acceleration of the respiration may depend upon several circumstances, and so also its slackening ; and special inquiry is necessary before we can be assured in any given case, that these are exactly opposed to each other. Analogy, at least, would suggest that atropine paralyzes the inhibiting respiratory centres,[1] and that the respiratory movements are thus accelerated in the same way as the cardiac, when their inhibitory apparatus is paralyzed. In antagonizing morphine, the same succession of events presents itself for the respiration as for the heart at the beginning of morphine poisoning. The respiration may be slowed, because the increased intracranial pressure has stimulated the inhibitory centre of inspiration, as it has the roots of the vagus and of the motor oculi nerve. Then the paralyzing effect of atropine would be beneficially antagonistic. Later on, when the susceptibility of the inspiratory centre itself is becoming benumbed, it might be (according to our theory) indirectly aroused by more rapid capillary circulation both throughout the tissues and in the medulla itself. By accelerating the circulation, therefore, atropine brings to bear upon the inspiratory centre the normal blood-stimulus to which it is physiologically adapted to respond. The antagonism to the effect of the morphine would therefore be indirect.

We would note, in passing, that the common assertion (which Dr. Bartholow endorses), that morphine induces carbonic acid narcosis, seems to us very inaccurate. The characteristic reaction of the inspiratory centre to an excess of carbonic acid in the blood is convulsion, which morphine does not cause in adults. We think it could be shown that the slackening of the respiratory movements coincides with, and follows, diminution of molecular respiration in the tissues. The phenomena are those of apnœa, not of asphyxia ; there is not an excess of carbonic acid irritating the inspiratory centre, but a deficiency, and leaving it in abnormally long intervals of repose. Hence might be suspected another mode of action of atropine, viz., accelerating the circulation and tissue-change. But into speculations like these, Dr. Bartholow

[1] Described by Rosenthal, Bemerk. üb. d. Thätigkeit d. automatischen Nervencentren, etc. Erlangen, 1875.

(even was the genius for limitations required, not only for power[1] but for scientific summaries) does not enter. His summary, however, contains many illustrations of the doctrine we maintain, namely, that effective antagonism is always either paralytic or indirect. Thus, having no direct control over the cardiac tetanus of angina pectoris, we can yet relieve the attack by paralyzing the contracted arteries through inhalations of amyl nitrite. Failing to arrest uterine hemorrhage by astringents directly applied to the bleeding surface, we may effect our purpose with nux vomica, which "stimulates the cardiac and respiratory centres."

And so on. The more examples we multiply the less should we be ready to accept Dr. Bartholow's doctrine of mutual antagonism by means of "opposed actions in the same tissues;" the more inclined to believe that the antagonistic influence is necessarily exerted upon different organs, or upon tissues in the same apparatus.

We have selected for comment the topic that happened to attract our attention. We leave to others the agreeable task of seeking food for other reflections from these most suggestive lectures. [M. P. J.]

Medical Electricity: A Practical Treatise on the Applications of Electricity to Medicine and Surgery. By ROBERTS BARTHOLOW, A.M., M.D., LL.D., Professor of Materia Medica and General Therapeutics in the Jefferson Medical College, etc. With 96 illustrations. Philadelphia: Henry C. Lea's Son & Co., 1881, pp. 262.

The announcement of a new work on medical electricity gives rise at once to the queries: Why has another been added to the numerous ones already published? What faults and deficiencies in the latter does the former correct and supply? for certainly a sufficient number of defective works on this subject are already before the public. In the preface to the above-named work the author gives an answer to these questions from his standpoint, namely: "That there are excellent works on medical electricity, is undeniable; but some of them are too voluminous, others too scientific, and not a few wanting both in fullness and in accuracy. I have attempted, in the preparation of this work, to avoid these errors; to prepare one so simple in statement that a student without previous acquaintance with the subject may readily master the essentials, so complete as to embrace the whole subject of medical

[1] According to a remark quoted by Lewes from, we think, Goethe.

electricity, and so condensed as to be contained in a moderate compass. I have assumed an entire unacquaintance with the elements of the subject as the point of departure, for I am addressing those who have failed to acquire this preliminary knowledge, or having acquired it, find that after the lapse of years it has become misty and confused." That the author has been quite successful in the accomplishment of his object few would deny. His pleasing style and clearness of expression cannot fail to make a readable book, even when applied to a dry subject. Yet lucid statements and attractiveness of style may fail to thoroughly instruct the reader. A few pages devoted to the enunciation of the simple laws which govern electrical phenomena, with a few illustrations of their application, will do more to instruct than whole chapters which describe such phenomena in the most simple language, but fail to go to the root of the matter and refer them to fundamental principles. While this applies to the work in question to a much less degree than to the majority of works on medical electricity, still it is not wholly inapplicable.

Our author states that: "In the account of electrical phenomena I have adhered to the modes of expression with which the medical electrical text-books have made us familiar. The time has not yet come, it seems to me, to adopt the terms and explanations now employed by practical electricians; it is a transition period in which both the old and the new should have a measure of recognition." It is disappointing to find, with this acknowledgment of the existence of new terms and explanations, an adherence in this new book to old modes of expression. If, as stated, the book is intended for the instruction of those who have not acquired such knowledge, and those who have forgotten that which they once knew about it, would it not be better to start them on a reformed basis than on one that is obsolete or retiring? Or, if both are to be recognized, let the modern one be put in the foreground as a working basis, thus saving the student from the difficult period of transition by making him familiar in thought with that which he must use sooner or later, while the older forms of expression may be easily explained and their defects pointed out.

Part I (pp. 80) is devoted to electro-physics. The chapter on "forms of galvanic combinations" gives a more complete account of the chemical reactions taking place in the different cells described than is usually found in works on medical electricity. It is surprising to find in Part II, on electro-physiology, in referring

to animal electricity of nerves and muscles, and the experiments of Du Bois Reymond and others, that no mention is made of the more recent experiments and views of Hermann ; although it is expressly stated that in consequence of the uncertainty of our knowledge on this subject only the slightest sketch would be given ; yet to have mentioned Du Bois Reymond's most powerful opponent in this great physiological war would have required but a few words, while it would have furnished the reader with a hint to further research.

In Part III, on electro-diagnosis, one of the most important subjects in electro-pathology is considered ; namely, the "degeneration reaction," a subject which has been very slow in finding its way into text-books in English on medical electricity. This is one of the best features of the work. It is quite remarkable, however, that our author should have included, without any qualification, progressive muscular atrophy, with glosso-labio-laryngeal paralysis and infantile paralysis, as examples of diseases in which the degeneration reaction is to be found, when it is well known that it is the exception, and not the rule, to find qualitative changes in the first-named disease ; and that reaction to faradism is usually preserved, though possibly diminished, in the muscles as long as there is a trace of muscular tissue left, this very point being of considerable diagnostic value. It may be that the true degeneration reaction always exists in certain fibres undergoing atrophy, but as the muscle is not affected *en masse*, it would not be easily recognizable, being masked by the reaction in the unaltered fibres until a very late stage. So that, practically, from a diagnostic point of view, this disease does not conform to those with which it is associated by our author.

In Part IV, on electro-therapeutics, we find a conservative view maintained as compared with the majority of text-books ; though not infrequently it seems as if exceptionally favorable cases had been selected, which are, no doubt, encouraging to the student, until he finds that failures, or imperfect results, are more frequent than the successes he had expected. A point not sufficiently appreciated is maintained by the author, namely, the necessity for *frequent* application of electricity, particularly in the treatment of painful affections ; for example, in cervico-brachial neuralgia he recommends "*séances* of five to ten minutes' duration, three times a day." It is questionable whether sufficient stress is laid upon the polar method in the treatment of painful affections. It is stated that "it is good practice to apply the anode to the painful

point ;" this, however, is rather luke-warm compared with the views of some acknowledged authorities.

Part V treats of electricity in surgery, presenting a good chapter on electrolysis. In the succeeding chapter on electric heating and lighting is an explanation of the "secondary cell" of Planté used in Trouvé's *polyscope ;* as justly observed "the principle involved is of great importance, and as it is likely to enter largely into the construction of medical electrical apparatus, the reader ought to have a clear comprehension of it and of the apparatus." After describing various forms of cautery batteries and their uses, also Adams's electric laryngoscope, the book is concluded by Part VI, in which thermo-electricity is considered, and reference made to Lombard's thermo-electric pile as an instrument for determining variations in body temperature. [W. R. B.]

Supplement to Ziemssen's Cyclopædia of the Practice of Medicine. Edited by GEORGE L. PEABODY, M.D., Instructor in Pathology and Practice of Medicine, College of Physicians and Surgeons, New York ; Pathological and Medical Registrar to the New York Hospital. New York : Wm. Wood & Co., 1881, pp. 844.

The object of this volume, according to its editor, is to give a concise account of the progress made in the various departments of medicine during the time that has elapsed since the several volumes of the cyclopædia were published. Only those subjects are treated which appeared in the American edition, and some of these are omitted ; nothing of importance having appeared relating to them. The space assigned for the review of this volume will not allow a list of its twenty-eight contributors, or the titles to the sixty-one subjects treated, to be given ; preventing entirely a review of the articles separately. Suffice it to say that eleven of the contributors are from New York, ten from Boston, three from Chicago, one from Philadelphia, one from Cincinnati, one from Ithaca, and one from the U. S. Army.

The work is, in the main, a bibliography, supplementary to that of the cyclopædia, and a review of the same. Some of the contributors, however, have treated the subject from their own standpoint, impressing the stamp of their own individuality upon their article, which, for the average reader, makes a more satisfactory production for perusal. Such a work must, of necessity, be more or less imperfect and unsatisfactory. The limitation of space, necessary to prevent the work from becoming too voluminous, often

cripples the reviewer and renders his summary incomplete in the number of articles referred to, or imperfect in his treatment of them. One of the most original and most valuable articles is that of Geo. Sternberg, M.D., U. S. Army, on Yellow Fever, as it sums up our knowledge concerning this disease after the experience derived from our recent epidemics, and the efforts, both individual and national, to elucidate this important subject. Coming from the pen of one who has devoted himself especially to this topic during the eventful period named, his article well deserves study. However unsatisfactory it may be to find that so many doubtful points are still left in doubt, we must feel gratified at seeing evidence of a conservative spirit in the conclusions given. It is surprising to find in reviewing this book how important a place the germ, or parasitical, theory of disease has occupied in the thought and work of medical men during the last few years; not limited, as formerly, to the realm of pure speculation, but largely devoted to reasoning based upon critically experimental data. Here, also, conservatism holds the balance of power.

One of the most valuable features of this work for the thorough student is its bibliography. It is to be regretted, however, that there is so much inequality in this particular. In the article on Syphilis, by Prof. James Nevins Hyde, of Chicago, the editor has left out altogether "a very voluminous and carefully selected bibliography, containing nearly five hundred references," on account of limited space. A carefully selected bibliography is of more importance, however, than a voluminous one. Indeed, unless the worthless abstracts and reviews, and the unimportant articles are excluded, a bibliography becomes a hindrance instead of a help to the student. On the whole, the work represents a large amount of bibliographical research, and no one who has not attempted such a task is likely to appreciate the consumption of time and the judgment required to collect and summarize such a mass of data as appears in this volume.

To the thinking reader it presents a vast array of facts and theories which proper study may utilize, while to the routine practitioner or the superficial student, it presents, in many of its chapters, a quagmire of irreconcilable views; in others, a field barren of that fruit which he seeks, namely: the strictly practical. In this respect, many of the contributors have been true to the original work which they have supplemented. To those who possess the cyclopædia this volume forms an indispensable addition, and even those who do not possess the former will find it a valuable acquisition by itself. [W. R. B.]

The Metric System in Medicine. By OSCAR OLDBERG, Phar. D. Presley Blakiston, Phila., pp 182.

As a book of reference the little work before us will be found extremely useful and accurate, but if intended to radically further the cause of the metric system among Americans it will share the fate of its predecessors for reasons which will be given later on.

Part first commences with some historical remarks. Among these we look in vain for a mention of the labors of the late Doctor Edouard Seguin, who by teaching and example has done perhaps more than any one American to popularize the metric system.

After a brief description of metric terms come 24 pp. of tables of equivalents of linear, square, and cubic measures and of weights, very accurate and of great usefulness for reference.

Next follows, occupying 77 pp., a metric prescription formulary, which contains three hundred and thirty-four formulæ, selected, the author states, "from the pharmacopœias and formularies of the great hospitals of New York, Philadelphia, Boston, London, etc., or are contributed from the practice of the medical officers of the Marine Hospital service, who have been using the metric system exclusively since April 27, 1878. Quite a number of the prescriptions are transcribed from the hospital formulary compiled by Chas. Rice, Ph. D., of New York."

Culled from so many sources it will be hard to trace the outrageous Latin they contain to its proper source. After a pretty careful perusal of the formulæ not one unabbreviated word was found in the genitive case. The following is a prescription appearing on page 119.

℞

Sulphur. præcip.,	15 gm.
Pyroleum cardinum,	15 gm.
Creta præparata,	10 gm.
Sapo mollis virid.,	30 gm.
Butyrum petroleum,	30 gm.

As the book is intended "especially for students" (preface) good Latin, if Latin there must be, should be placed before them.

As the formulæ are introduced simply to illustrate the way of writing metric prescriptions it would seem as if three would do as well as three hundred.

Next come dose tables occupying 53 pp., very full and presumably accurate, although we see among them the dose of fluid ex-

tract of conium to be only .1—.4 (2—5 minims), and that of iodide of potassium to be .1—1. (2—15 grains).

Now to return to the first part of the work. The title of the book tells us the work is "an account of the metric system of weights and measures Americanized and simplified," etc. To simplify the metric system is impossible, it being already the most rational and simple. To Americanize the system the author says (p. 20) "we will hereafter drop the term cubic-centimeter, and adopt in its place the term fluigram. . . . . the word fluigram is, besides, more convenient, euphonious, and American than the word cubic-centimeter." Again, p., 23, "Tenths, hundredths, and thousandths of both the gram and fluigram may be conveniently called 'dimes,' 'cents,' and 'mills' when referred to in speaking." Now, we claim that to Americanize the metric system in any way, except to spread it broadcast over the country in its original form, is to destroy its most commendable feature, *i. e.*, that of international uniformity. If we adopt the metric system, let us adopt it entire, French being much more universally understood among scientists than English. Such slight deviations from the original as cubic-centimeter for centimetre cube and gram for gramme, being unimportant.

The abbreviation gm. for gram we think injudicious, as it is often mistaken for our old gr. We think the decimal point or line, as advocated by the late Dr. Seguin and used by him in his Prescription and Clinic Record, quite sufficient, more simple, and less liable to permit mistakes.

For example, Prescription No. 216, p. 100, reads :

℞

Ferr. reductum,	7 gm.
Quinin. sulph.,	8 gm.
Strychnin. nitr.,	0.15 gm.

This, it seems to us, is less simple and not so safe in the druggist's hands as :

℞

Nitrate of strychnine,	0.15		0	15
Reduced iron,	7.00	or	7	00
Sulphate of quinine,	8.00		8	00

neither of which could be well taken for any thing but a metric prescription.

"To drop the old system of weights and measures entirely, and start out anew with the metric system, after learning the doses of medicines over again in metric terms, I conceive to be more con-

venient than safe," p. 17. We think it would be more safe than convenient, if by convenient he means easy.

To popularize the metric system we must begin with the student. To whom shall we look for help? Most assuredly to our writers of students' text-books, to our medical journals, and to our didactic and clinical lecturers. What are our authors doing to further the cause of the metric system? Take the three most popular works in America on materia medica and therapeutics: Wood's, Bartholow's, and Stille and Maisch's. In all the doses in the text are given in grains and minims; in one only is a posological table in both systems; in another there is a table of equivalents hidden in an appendix at the end of the book; while in one not a metric word or figure occurs in the whole book.

Few medical journals in our country uses the metric system exclusively, and the number of our lecturers so using it could be counted on the fingers of one hand. [R. W. A.]

Anatomical Plates. By Prof. J. N. MASSE, Paris. Arranged as a companion volume for "The Essentials of Anatomy." Edited by AMBROSE L. RANNEY, A.M., M.D. G. P. Putnam's Sons, New York, 1881.

For many years Masse's Anatomical Plates have been known to English readers through its translation by the late Prof. Granville Sharp Pattison. This translation is assumed by the editor of this volume; and, having made such alterations and additions as were required to bring the plates and text up to date, he offers it to the profession as a companion to his "Essentials of Anatomy."

The book illustrates osteology, arthrology, aponeurology, splanchnology, myology, angeiology, and neurology. To these illustrations have been added diagrammatic cuts of many nerves, with special reference to their communications and distribution. As illustrations of anatomical fact the plates are accurate and well executed, and their arrangement relative to the text renders reference as easy as possible.

For the purposes, however, for which this work was prepared, we prefer the use of color and greater size, thereby to secure a more immediate perception of an organ and its relations. For the "country physician and surgeon," for whom this and so many other reference books are kindly made by the "brothers in town," there is the greater necessity that reference should be ready and easy. A paged index would help in this respect, and when the plate desired is found it should represent as many of

the sought-for relations of an object as are possible in a picture. Suppose, for example, a "country surgeon" should be called on to ligate the femoral artery, and, in the matter of anatomy, should consult this book, he would find in one plate the artery in relation with muscles ; in another plate, the artery in relation with the nerves ; while its relations with the veins, and the not-a-little important sartorius muscle, are not shown at all. These matters may have been left to the text-books. We are tempted to select an example of the insufficiency of the plates for the physician ; because, it may be, the editor assures us in the preface, that when the physician "wishes to refer to the position of any particular viscus, and to study its relations, or when symptoms depending on nervous connection arise in disease which he cannot explain, referring to this atlas, all his difficulties are removed." This quotation may mean and promise more than the writer of it intended. In truth, we are constrained to remark the same of the whole preface.

We would note a minor defect in the putting-together of the book. Some half dozen of the plates are inverted, so that it becomes necessary to turn the book around during reference.

The book is of moderate size, and its cost is brought within the means of every one in need of it, and, as a remembrancer, it may meet most of the reasonable demands made of it.

[J. V. D.]

ORIGINAL OBSERVATIONS.

TUMOR OF THE MOTOR ZONE OF THE CEREBRAL CORTEX.

By CHARLES K. MILLS, M.D.,
NEUROLOGIST TO THE PHILADELPHIA HOSPITAL.

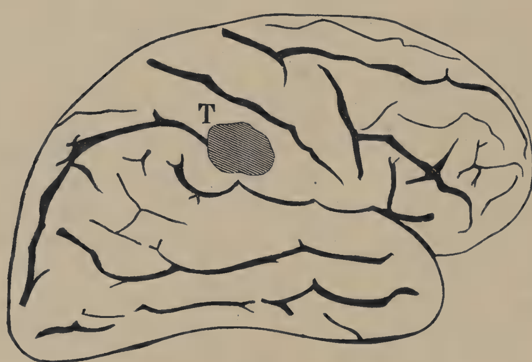
In August, 1880, I saw, with Dr. F. Dercum, of Philadelphia, the following interesting case :

Mrs. W., aged about 30, in the autumn of 1879 began to suffer with headaches. In March, 1880, she had an attack, beginning with numb sensations in the fingers of the left hand. These were followed by twitching movements of the fingers. The spasm extended to the left arm, and before the attack passed off a general convulsion occurred, the movements being most violent on the left side. After this seizure she found that the left upper extremity was decidedly weaker than the right. In May, 1880, she had another spasm, which involved only the left upper extremity. Subsequently, up to the time that she was first seen by me, she had about half a dozen more spasmodic attacks, which began with twitching movements of the fingers of the left hand. The convulsion was always most severe upon the left side, and was usually limited to it. Its greatest violence was spent upon the arm. After each attack the left half of the body became more and more paretic. The left upper extremity showed the greatest amount of paralysis. She suffered more or less pain in the head all the time, and at frequent intervals had paroxysms of agonizing pain, accompanied by vomiting.

In September, 1878, Dr. Wilson Buckby had attended this patient for typhoid fever. I learned from Dr. Buckby that on the fourteenth day of her sickness she had had a severe convulsion, on the subsidence of which she was left with partial paralysis of the

limbs and face of the left side. In four days this paresis disappeared, and the fever ran its regular course to recovery. She had no other attacks of spasm until March, 1880.

In August, 1880, her condition was as follows : Her mind seemed clear, but acted slowly. She answered correctly, but not quickly. It was difficult for her to fix her attention. She would frequently burst into tears, apparently because of her excruciating headache. The pain was worse in the right fronto-parietal region. Percussion above and around the right ear caused greater pain than at any other place on the head. Sight was very imperfect, and ophthalmoscopic examination showed double optic neuritis. Hearing was defective in the right ear. Sensibility was impaired, but not abolished, on the left side. Both the upper and lower portions of the left side of the face were partially paralyzed. Paralysis of the



left arm was nearly complete ; the limb was a little wasted, but showed no contractures. The same condition, but less marked, was shown by the left lower extremity. The left patellar reflex was diminished. No aphasia was present. The bowels and bladder were partially paralyzed. The urine contained neither albumen nor sugar.

The patient died August 27, 1880.

A *post-mortem* examination was held 37 hours after death, Drs. Dercum, Buckby, Collins, and myself being present. Beneath and adherent to the pia mater of the convexity of the right hemisphere was found a tumor about 1½ inches in diameter. It was nodulated. On section, it was bloody, and had a mottled appearance. It involved the middle portion of the ascending parietal convolution and the upper part of the inferior parietal lobule, pushing

aside the interparietal fissure. The anterior extremity of the pupil was about $\frac{1}{5}$ of an inch back of the centre of the fissure of Rolando. On the inner side of the tumor the white matter of the brain was broken down. The only other lesion discovered was a slight adhesion of the dura to the pia mater over the upper extremity of the ascending convolution of the left side.

The tumor was examined, microscopically, by Dr. L. B. Hall, of Philadelphia, who reported as follows: "On section of the hardened tumor left with me, I found it to be of the same brown color throughout. The microscope showed this color to be due to numerous very minute points of hemorrhage scattered throughout the entire mass. The cell element consisted of large, rounded, multinuclear cells, filling a stroma of very fine fibres, with relatively large interspaces. Retrograde changes appeared in places where the whole was little more than a diffluent mass of debris. The appearances of the specimens best agree with carcinoma cerebri."

Remarks.—A considerable number of cases similar to the one here reported are now on record. The case is one which illustrates the possibility of making an accurate local diagnosis of tumors involving the motor zone of the brain and immediately adjacent parts. Before the death of the patient I expressed to Drs. Dercum and Buckby the opinion that the case was one of tumor of the cerebral cortex, involving the middle portion of the ascending parietal convolution and the adjoining parieto-temporal region. I further stated my belief that the growth of the tumor had probably begun in the ascending convolution. The distinctive general symptoms of brain tumor were present, namely: agonizing headache, vomiting, vertigo, psychical disturbances, and optic neuritis. Certain symptoms, to my mind, pointed conclusively to a tumor of the motor zone, and one primarily implicating the brachial centres. These were (1) the occurrence of spasms, beginning invariably in the fingers of the left hand, sometimes limited to the left upper extremity, and always either limited to, or most violent upon, the left side of the body; and (2) the occurrence of paresis, and eventually of marked paralysis, of the left arm, leg, and left side of the face, the paralysis of the left arm being the most complete. The involvement of the parieto-temporal region was indicated by the impairment of sensibility and of the special senses. The localized headache and the pain elicited by percussion above the ear, confirmed the localization indicated by the other symptoms.

REPORT OF TWO CASES OF INTRA-CRANIAL DISEASE.

By WM. S. CHEESMAN, M.D.,

FORMERLY HOUSE PHYSICIAN TO BELLEVUE HOSPITAL.

The following cases have seemed to me worthy of record in the interests of cerebral localization :

CASE I.—Bridget R., æt. 42. No history of injury or syphilis. Three years before her admission to the hospital she began to have slight spasms of the right side of the face, at irregular intervals, and infrequently, not oftener than once a month. Two months before admission, these convulsions became very frequent, occurring every few minutes, and about one month later the right upper and lower extremities began to participate in them. During the convulsion consciousness was not lost, and the intervals were free from symptoms. She had no pain at any time. Sight, hearing, and ocular movements unimpaired.

Two days before her admission, the patient became unconscious during one of these attacks, and, on recovering consciousness, was found to be hemiplegic on the right side. She had right unilateral convulsions every few minutes, and touching the right side caused her to cry out.

When admitted she answered questions rationally, though her speech was thick. Her replies were often interrupted by a right unilateral convulsion, during which she would lose consciousness. Right hemiplegia, with conjugate deviation of the eyes to the left, existed in the intervals. Pupils normal and sensation unaffected. Albuminuria and hyaline casts. No cardiac lesion. Temperature 100° F.

On the third day after admission the patient died. Her symptoms were considered to depend upon a cerebral tumor located in the cortex of the left hemisphere.

Autopsy.—On viewing the brain after its removal, a bulging was noticed on the left hemisphere. On longitudinal section of the left hemisphere, the medullary substance was found reddened, and the line of the cortex very indistinct. To the touch the brain was almost diffluent. A portion of the hemisphere, three inches long, and one and a quarter inches deep, was thus affected. The ascending frontal and ascending parietal convolutions and a part of the third frontal convolution were implicated, the disease extending inward to the lateral ventricle. The contrast between the healthy brain tissue of the posterior lobe and the soft, mottled portion affected by disease was very marked.

Macroscopic appearances at the time of the autopsy seemed to indicate that the lesion was an acute cerebral softening, but the microscope showed it to be a glioma.

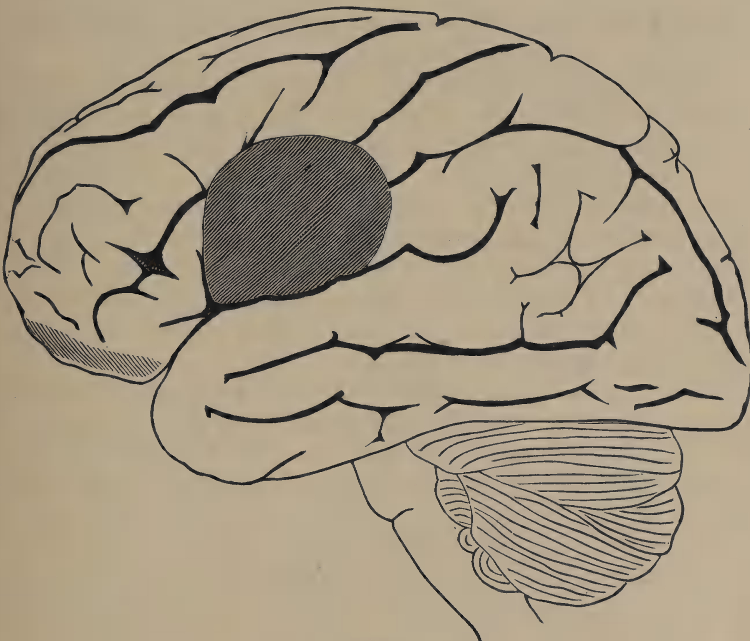


FIG. I.
External extension of glioma in Case I.

CASE 2.—Mary Ann W.

The patient was brought to the hospital comatose. No history could be obtained. No signs of injury to the head. Pupils normal. Temperature, 101¾°. No albuminuria or casts. No cardiac lesion.

In the evening, after admission, the patient had a right unilateral convulsion, limited to the face, neck, and upper extremity. These convulsions continued through the night at frequent intervals. The patient died next morning with pulmonary œdema.

In view of the symptoms it seemed probable that she had suffered from some lesion of the cortex of the left hemisphere, affecting the motor centres of the face and upper extremity; though, in the absence of history, the nature of this lesion could only be conjectured.

Autopsy.—No fracture of the skull could be discovered, and no signs of injury. But, on removing the calvarium, a clot was found covering a portion of the surface of each hemisphere. The brain substance beneath was softened, but the rest of the organ healthy.

The areas affected were :

On the *right* hemisphere, the upper extremity of the ascending frontal convolution ; on the *left* hemisphere, the upper extremity of the ascending frontal and the posterior extremity of the first frontal convolution.

FIG. 2.

Convexity of the brain after Ecker. Areas of clots and injured cortex.

The softening was less marked on the right than on the left side.

The clot on the right side had caused no symptoms.

A SOMEWHAT REMARKABLE CASE OF GLAUCOMA—APHASIA —DEATH FROM PROBABLE APOPLEXY.*

By DAVID WEBSTER, M.D.. New York.

Mr. N. B. first consulted Dr. Agnew in the spring of 1863, for a gradual impairment of vision in his left eye. The eye was found to be affected with chronic glaucoma, and an operation was advised. When the patient came for advice a second time, in the spring of the following year, the eye had lost all perception of light and was very painful. In short, the disease was *glaucoma absolutum.* The patient was then ready to assent to any thing for the relief of his pain, and Dr. Agnew performed an iridectomy upward with the result of quieting the eye. He had no more pain or inflammatory symptoms in this eye to the day of his death.

March 15, 1866.—Mr. B., now æt. 44, and married, is apprehensive of loss of sight of right eye. Reads J. No. 1, but sometimes sees better than at other times. Has a small crescent of choroidal atrophy at temporal border of optic disc.

Nov. 28, 1871.—Patient complains that a haze came over right eye while he was reading the newspaper last evening, and continued for about five minutes. Vision $= \frac{20}{30} +$. Ophthalmoscopic examination shows some excavation of the optic papilla, and pulsation of the retinal veins. Tension $+$?

May 27, 1873.—Patient has had several periods of slight obscuration of vision of late. Vision $= \frac{20}{20}$; no limitation of visual field. Tension $+$? Ophthalmoscopic examination shows precisely the same appearances as noted under last date.

March 18, 1874.—The patient being very anxious about his eye, was sent to Dr. H. D. Noyes in consultation. Dr. Noyes carefully examined Mr. B., and wrote as follows: "Mr. B., I find, has R. V. $= \frac{20}{30}$, with no impairment of field. Tension $+ 1$, or perhaps more. With $+ \frac{1}{3}$ I see molecular opacities in the lens, but none in the cornea or vitreous. The nerve exhibits a shallow central excavation, veins ampulliform and pulsating, arteries very small and emptied on slight pressure. The vessels follow the line of the pit in a way which makes me think that this is not due to original excavation, but to the pressure. I should favor an iridectomy with little delay, because of the existing effect on the arteries and of the changes in the lens. I do not think the risk will be important. As to the fellow eye, I see no reason to enucleate, because it is not troublesome, while if

* From the practice of Prof. C. R. Agnew, M.D.

it were I should rather do an opposing iridectomy, believing that would be sufficient. Unless a glaucomatous eye is painful I would not remove it to protect the other, and as such is not here the case, I would try to save the organ."

It was decided, however, to defer the operation until there should be more marked impairment of the vision, or limitation of the visual field, especially as there was a peculiar hebetude in the behavior of the patient that suggested the propriety of more than usual caution on the part of the surgeon.

June 1, 1874.—Some arching forward of iris from commencing proliferation of vitreous; venous pulsation more marked, and slight pulsation of one branch of the central retinal artery. Vision $= \frac{20}{20}$; visual field remains normal.

Sept. 1, 1874.—Called to see Mr. B. in consultation with his family physician. He was, some days ago, attacked with vomiting and severe headache, referred to the left fronto-parietal region. At the same time he became the subject of aphasia and agraphia, being unable to express himself either orally or in writing. To every question he would reply, "You see," or "You know." Pulse 60, temperature normal. His family physician has learned that he had syphilis some twenty years ago. Put upon iodide of potassium in increasing doses.

Oct. 10, 1874.—Patient has sufficiently recovered to come to the office. He seems to be slowly recovering from his aphasia. The question was asked: "Have you any pain in the head at all?" He replied, "Yes, across here," puting his hand on the top of his head. "I feel a kind of a treatment across there—I can excel, I can try a good deal of people. I can tell a good many, that is, people of the past, in the time. My treatment here I could pass. Am well."

Question.—"Can you write?"

Answer.—"Yes, pretty well."

"Write." Patient writes:

"N. B. hot aurred baths with gunds every week—Unicas St."

Dr. E. C. Seguin, who saw Mr. B. on October 15th, writes as follows:

"Your patient, Mr. B. has a peculiar form of aphasia, or, more properly speaking, he is in a very peculiar stage of recovery from aphasia. He is reconstructing his language and the attempt is strangely incoherent. There being no cardiac murmur, I incline to the opinion that he has had thrombosis of a small branch of the left Sylvian artery, supplying Broca's region. He

admits having had venereal disease, but is in no state to answer questions as to the particulars of the attack : the thrombosis may have been the result of syphilitic arteritis."

Mr. B. died about six months after his attack, having never completely recovered from the aphasia. There was no *post-mortem* examination in his case, but the certificate of death records, "softening of the brain."

The case is an interesting one in many respects. The fate of the left eye shows how dangerous it is to defer an operation in chronic glaucoma, where the sight is rapidly failing. Such cases are almost certain to terminate in a painful inflammatory absolute glaucoma, and when this condition is reached an iridectomy does not always relieve the pain, as it happily did in this case, but very frequently such eyes have to be enucleated.

The history of the right eye shows that the prodromata of glaucoma may exist for years without marked impairment of vision or limitation of the visual field. The event shows that it was just as well that the operation was not performed upon this eye, as the vision remained very nearly perfect, except during short periods of obscuration, as long as he lived.

If there was any causative relation between the disease of the eyes and the "softening of the brain," it is difficult to explain it. Syphilis was probably the cause of the intracranial lesion, and it may, indeed, have been the primary cause of the glaucoma, but of that we are by no means certain.

HISTORY OF ATTEMPTS MADE TO CURE THREE CASES OF CHRONIC TRIGEMINAL NEURALGIA.

By E. C. SEGUIN, M.D.

It must have appeared to many physicians besides myself that the custom of reporting only successful cases, and of slighting, or altogether omitting, an account of our unsuccessful attempts at cure, was a bad one, and this for several reasons. One of these is that the perusal of such one-sided reports is quite sure to inspire some of our *confrères* with undue confidence in the power of drugs over disease, and to shape their prognosis accordingly.

Among the diseases which most tax our patience and therapeutic skill, there are few more redoubtable than chronic trigeminal neuralgia, or tic douloureux. Excellent as is the reputation of this affection for incurability, yet the published records of

this committee embrace several instances of its cure by drugs in patients who had suffered fourteen years or less (*vide New York Medical Journal*, Dec. 1878, p. 621).

I propose this evening, for the purpose of enabling you to profit by my unsatisfactory experience, to relate briefly the history of three cases of the disease in question, which have not been cured.

CASE I.—Mr. F. O., aged 45, oyster dealer. History taken when first seen, Dec. 12, 1878. General health has always been excellent. In 1856 had a single malarial chill, followed by two slight attacks of right supra-orbital neuralgia.

Present tic douloureux began in 1857, by a few "sticking" pains near the right infra-orbital foramen : a single pain like the pricking of a needle several times a day. This pain steadily increased in severity and frequency. Came north from Georgia in 1858, and for one year was free from pain. After that time it returned. Two or three times a year afterward he had spontaneous relief for some weeks. In the last two or three years constant suffering. Patient has tried a good many medicines without relief.

Now has a paroxysm of pain every two or three minutes day and night. Eating, drinking, talking, attempts to wash or wipe the skin of the face on right side excite paroxysms of pain.

About three years after commencement of trouble (1857) the pain extended to the whole of the upper maxilla, later to the lower jaw, and recently the whole of the right trigeminus, lingual branch included, has been the seat of pain. There is no regularity or periodicity in time of appearance of the pain, or in its degree of intensity. The patient never has common headache or dizziness. In 1857–'58 one tooth was pulled from the right upper jaw, and another in 1867 ; pain aggravated each time.

Denies injury to face and syphilis.

Examination.—Patient is a large and powerful man, of healthy aspect, with a facies indicative of suffering. Every few minutes he has an epileptiform (*i. e.*, sudden) onset of pain in right side of face and head to vertex ; pain sharp and cutting ; paroxysm lasts a few seconds, and during it the face flushes. The cutaneous sensibility of the affected region is normal to simple touch and to æsthesiometer test. Hearing of right ear O, drum thick and whitish. Hearing of left ear 12–15 inches (watch). The corneæ are normal ; right pupil is a trifle smaller than the left in intervals between pains. The teeth on right upper and lower jaws are

covered with an extraordinary layer of "tartar," and some are loosened. Patient has not dared cleanse teeth on that side for years. There are no tender points upon the face or in the mouth. Teeth on the left side are fairly clean.

Was ordered solutions of Duquesnel's crystallized aconitia, in doses of $\frac{1}{60}$ grain, and this was given in increasing doses, with no relief. On Dec. 18th, following note recurs: Aconitia must be deemed a failure. Has taken $\frac{1}{10}$ grain in 24 hours. Constant great effects on sensory nerves, coldness and tingling. Has pains almost every two minutes. Fowler's solution ordered in increasing doses after meals. Dec. 30th. Has increased arsenic to 16 drops after each meal; nausea; no relief to pain.

Ext. gelsemii fluid. ordered Dec. 30th, gtt. v before each meal, and at bedtime. Jan. 14 (1879), full effects of gelsemium obtained from doses of gtt. xiv and xv, four times a day. No relief to pain.

Sol. phosphori Thompson (ʒ i$=\frac{1}{19}$ grain P.), tried in doses of ʒ i an hour before each meal for several days; no effect.

Injections of chloroform in cheek used on Jan. 20th, 21st, and 22d. Injections made through mucous membrane, toward right infra-orbital nerve. Five minims on 20th, ten on 21st, with no relief; slight swelling and burning pain. Attempt to inject m. xv on 22d resulted in asphyxia, and apparent death, previously reported to the committee.

Mixed treatment, iodide of mercury, and saturated solutions of iodide of potassium ordered on Jan. 23d. On Feb. 11th slight effect on gums is noted; takes about 40 gtt. of sol. sat. K I. three times a day; no relief.

Galvanism, stabile, strong current (25 cell); kathode on tender points from 7 to 15 minutes. Patient thinks pain is aggravated by the current.

Ammonio-sulphate of copper ordered, .08 with ext. cannabis ind. 03. before each meal since Feb. 11th; stopped on 15th; no relief.

Operation.—Resection of right infra-orbital nerve performed, Feb'y . . Nerve removed outside and inside orbit. Healed by primary union.

March 9th. Face perfectly healed; only part that is absolutely anæsthetic to faradic current by brush in a spot about 2 cent. square under right eye. Has partial sensibility to brush, and pricking in rest of cheek, in ala nasi, and upper lip, and inner aspect of cheek and mouth. To-day less pain, but he suffered

very much on 6th, 7th, and yesterday. A paroxysm seen in office seems less severe than those before operation. Ordered quinia sulph. .25; morphia sulph., .02 three times a day.

March 14th. Much better. Few attacks in supra-maxillary region. Talking and chewing can be done without agony. Has had several severe attacks of pain in infra-maxillary region, and in outer part of orbit; not in supra-orbital district. Has had good nights. Continue quinia and morphia.

March 19th. Is fifty per cent. better than before operation (patient's own estimate).

March 28th. No "neuralgic" pain in right upper jaw and lip, but the lower jaw and lip are seat of severe neuralgic pains, not as severe as formerly. Ordered pil. quiniæ et morphiæ et belladonnæ twice a day. Ordered fluid extract of aconite, gtt. i *t. i. d.*

March 31st. No neuralgic pain in upper jaw; severe in lower jaw. Continue aconite.

April 14th. Considers his condition improved at least 50 per cent. Takes iv or v gtt. aconite, with slight physiological effects. Sleeps soundly. No severe paroxysms in two weeks.

During May more pain; severe paroxysms in anæsthetic district. Fowler's solution, aconite, morphia again tried in vain.

Was not seen again until Dec. 17, 1880. Was free from extreme suffering for several months. In last few months almost constant severe pain.

I have since tried aconitia and gelsemium to physiological effects, without relief.

Dr. Weir is planning to remove Merkel's ganglion.

CASE 2.—Mr. H. S., janitor, aged 29 years. History of case taken October 2, 1878 (*vide* a partial report on the case in *New York Medical Record*, Jan. 4, 1879).

Previous to the development of the present affection, he had been subject to occasional dull headaches. Ten years ago (1868) he suddenly experienced a very severe sharp pain all through his head, "as if devils were at work there," lasting half an hour. There was no dizziness or faintness, or nausea, or impairment of sight, or paralysis. For a period of six months he remained free from pain, and, indeed, was perfectly well. After that time, nearly ten years ago, a "dull, stupid pain" began over the right eye, extending from the supra-orbital notch inward to the nose, and down the side of the nose to the ala nasi. This pain was paroxysmal, and worse in the daytime. Later the pain extended to the eyeball, and was exceedingly severe, the paroxysms re-

curring from ten to twelve times a day. In the course of two or three years the pain made its appearance in the right temple—worse at night.

In the last few years most of the pain has been on the top of the head, above the temple, and in front of the ear to the bregma. There has lately been only an occasional pain in the side of the nose, and not much pain in the temple proper. During the past summer, and since, there has been some occipital pain on both sides—more on the right. In the last year there has also been pain in both the upper and lower jaws, in the upper lip near the median line; none in the tongue (on right side). In the last four years vision has been dim, and glasses have not remedied the defect. Five years ago had temporary diplopia, but this was while taking some unknown medicine. At various times during this long illness has had "dizzy spells," with varying frequency; few in the last months. Has had no other symptoms of a neuralgic nature. Memory is impaired and virility quite lost. Had severe dyspepsia and vomiting three years ago, and has been costive during the whole period of the disease.

Examination.—The various painful regions are hyperalgesic, but not numb, and the tactile sensibility is perfectly preserved on both sides. There is no facial paralysis; the right pupil is positively small, the left normal. After dilatation by atropine the ophthalmoscope reveals no lesion in the fundus. Hearing, smell, and taste are normal. Cornea clear. The urine (frequently examined by other physicians and found normal) is now free from albumen. Marked anæmia is exhibited by the skin and mucous membranes; has always been pale. Denies syphilis.

The pains, which occur frequently in my office, are the most terrible which I have ever witnessed; the patient fairly writhing in his chair, or even falling to the floor (not unconscious) in his agony. During the attack the right eye is much injected, and tears flow freely from it, while the left eye remains dry.

The patient states that no medicine has ever relieved him, and that he has tried a great many.

The treatment in this case, though prolonged until now, Feb., 1881, has been relatively simple.

Duquesnel's aconitia in doses of $\frac{1}{100}$ grain. Solutions by Neergard at first, later in the shape of Schieffelin's granules, given from two to four times a day. Full physiological effects were easily obtained, and were kept up for many months. Numbness and a remarkable cold chilly condition were the signs. At times the

subjective cold was so great that he would come to my office shivering in an overcoat.

In this case as in Case 3, increased susceptibility to the action of the drug was observed as time went by. In the last few months, one dose of $\frac{1}{100}$ grain produces effects which last from six to nine hours.

Besides aconitia, iron and Fowler's solution in moderate doses have been administered frequently. Has had several attacks of subacute rheumatism rapidly cured by sodium salicylate.

On the whole the result obtained is very gratifying—it is a relative cure.

Patient a few weeks after beginning of treatment experienced no excruciating paroxysms, and gradually resumed his occupation as janitor. In last few months seldom loses half a day. Has kept a record of attacks, classifying them into severe and mild : has had very few severe ones in each month, and has registered many days without any pain.

There has occurred a curious shifting of pain. It was formerly more intense in fronto-temporal region, it is now developed mostly near the parietal eminence.

The patient's general condition has greatly improved ; he still has a peculiarly white skin, but his lips, etc., are fairly well colored.

The change in *moral* is most remarkable ; is now cheerful and enjoys both his work and his family pleasures ; whereas about a year ago he looked upon life as a burden.

Case 3.—Mr. W. L. P., clerk, age 54 years, seen September 22, 1880.

Had always enjoyed good health.

In 1876 there appeared a pain in front of the right temporo-maxillary articulation ; a deep pain. At first the pain was occasional, excited by washing face. Pain has steadily increased in frequency and severity, until now paroxysms occur almost every moment. The pain is rather worse in afternoon and night, not typically nocturnal. In about a year after beginning the pain extended to infra-maxillary and infra-orbital nerves (never appearing at mental foramen). It extends into the gums in right upper and lower jaws, and "strikes" in the lower jaw at a point a little posterior to the angle of the mouth. No pain above zygoma and orbit. Saliva flows in the paroxysms. All movements of jaws cause more pain. Weather is without influence.

No malarial fever since his 16th year. Never had syphilis.

Used much tobacco until recently. Temperate. Has had seven teeth pulled from the right upper jaw without relief.

Examination.—No tender point except at the mental foramen, where there is no pain. No evident anæsthesia. Some atrophy of fatty tissues of face on the right side. Opening mouth causes a paroxysm. Hair on face kept stubby and is worn on cheek by constant friction of hand and fingers during paroxysms. Attacks last from one to one and a half minutes. General health good.

The treatment was begun Sept. 22d, by giving Duquesnel's crystallized aconitia, in the shape of tablets made by Caswell & Hazard, $\frac{1}{200}$ grain every two hours.

25th.—No strong aconitia effect. Sleeps without chloral. Ordered $\frac{1}{200}$ grain every hour. To-morrow $\frac{1}{100}$ grain every two or three hours.

27th.—Great relief; did not feel aconitia much, $\frac{1}{100}$ every two hours till 5 P.M., when he was quite numb, and sight was dim.

29th.—Marked improvement; pain only in zygomatic region. From the 29th to Oct. 1, included, sol. phosphori Thompson was used, ʒ i three times a day. Pain made worse. Again given $\frac{1}{100}$ gr. aconitia.

Oct. 4th.—Severe pain; no aconitia for one day. Takes sol. Fowler., gtt. viii after each meal, increasing. Ordered continue Fowler, and take ext. gelsemii fld. gtt. v every 2 hours. Continue and increase the Fowler's solution.

10th.—Ext. gelsemii fld. is also being used, but no aconitia. Takes gtt. viii of gelsemium every three hours with moderate effect; double vision at times; lids heavy. Very little severe pain; has lost habit of rubbing cheek in paroxysms; good nights.

20th.—Has reached a maximum dose of gtt. xvi Fowler after each meal. Gelsemium as above. The gelsemium is stopped; Fowler's continued, and aconitia, $\frac{1}{100}$ gr., every two hours ordered.

23d.—Very little pain in last forty-eight hours; feels the aconitia; attacks slight; pain nearly localized near right temporo-maxillary articulation; can eat and talk with little pain.

Iodide of potassium, saturated solution in doses of gtt. xx before each meal in much water, increased by 5 drops daily, was begun on 28th. Fowler's abandoned. Aconitia $\frac{1}{100}$ gr. *p. r. n.*

Nov. 3d.—Coryza and hoarseness, neuralgia slight. Takes gtt. xl. *t. i. d.* Stop. Cautery on focus of pain in front of ear tried on 5th. Pain aggravated.

Ammonio-sulphate of copper was tried during November, De-

cember, and January (1881), given in pills, dose increased from .05 *t. i. d.* after meals, to .20 after each meal and at bedtime. Aconitia $\frac{1}{100}$ was used *p. r. n.* by patient all the time.

Pain very variable; a few days almost without pain; other days much pain, often under influence of storm or rain. At one time copper *before* meals produced griping and watery stools; no ill effect when administered after food.

Dec. 11th.—The note is made that patient has become much more sensitive to aconitia; is affected in ten minutes by one tablet, whereas formerly it required an hour or more to obtain any prickling.

The whole of January, 1881, was very comfortable.

1st.—No severe paroxysms. Was in Canada part of the time. Now can use only one or two tablets of aconitia a day—formerly could take one ($\frac{1}{100}$ gr.) every two or three hours.

Feb. 19th.—In last month gelsemium and aconitia. Much more pain in last fortnight, though not as much as before treatment. Pain is severe in spite of full effects of gelsemium, gtt. v every two hours.

A fair summing up of these attempts at relief of incurable conditions is, it seems to me, that aconitia is the chief agent to be relied on for the alleviation of the pain of chronic trigeminal neurraliga, and for its cure. Of course, malarious and syphilitic neuralgias are excluded from this statement; in them we have special indications.

Gelsemium and arsenic have both seemed to exert a secondary beneficial influence.

Galvanism, the actual cautery, injections of chloroform, were useless. Morphia and chloral afforded mere temporary relief.

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ARCHIVES OF MEDICINE.

Original Articles.

THE INFLUENCE OF BAROMETRIC CHANGES UPON THE BODY IN HEALTH AND DISEASE.

By ANDREW H. SMITH, M.D.,
NEW YORK.

THE effect upon the body, in health and disease, of variations in barometric pressure, is a subject which has not received the attention from the profession which its importance entitles it to. Beyond some studies of the effect of altitude upon phthisis and the influence of barometric changes in determining pulmonary hemorrhage and attacks of spasmodic asthma, but little discussion seems to have been excited. Even the introduction of compressed and rarefied air as therapeutic agents appears to have done very little to stimulate inquiry as to the part played by the constant natural changes in the density of the atmosphere in preserving health or inducing disease. Yet these changes cannot be without their influence, and there is opened here a wide field, not only for speculation, but for scientific observation, which may bring important accessions to our knowledge of the etiology of those affections which appear in the form of attacks recurring at irregular intervals.

The introduction, within a comparatively few years, of the use of compressed air in submarine engineering opera-

tions, has given an opportunity for studying, on a large scale, the effect of a prolonged sojourn in a greatly condensed atmosphere, and of the subsequent removal of the pressure. The facts observed are very suggestive, and point unmistakably to a disturbance of the normal distribution of the blood with each change in the pressure of the atmosphere.

As the result of exposure for several hours to a pressure of two or three atmospheres, there may be developed a group of morbid phenomena to which the writer, in an essay published some years ago,[1] attached the name of the Caisson Disease. The definition of this disease is as follows:

A disease, sometimes fatal, depending upon increased atmospheric pressure, but always developed after the pressure is removed. It is characterized by extreme pain in one or more of the extremities, and sometimes in the trunk, which may or may not be associated with epigastric pain and vomiting. In some cases the pain is accompanied by paralysis more or less complete, which may be general or local, but is most frequently confined to the lower half of the body. Cerebral symptoms, such as headache, vertigo, and coma, are sometimes present. The above symptoms are connected, at least in the fatal cases, with congestion of the brain and spinal cord, often resulting in serous or sanguineous effusion, and with congestion of most of the abdominal viscera.

That such decided results, including even death, may be brought about by extreme changes of atmospheric pressure, certainly leaves room for the surmise, that slighter changes, occurring from natural causes, may produce, at least, proportionate effects. I think that the essay already referred to, contained the first suggestion, that this might be the explanation of the neuralgic pains which many persons complain

[1] Essay on the effects of high atmospheric pressure, including the caisson disease. Published by the East River Bridge Company, 1873.

of at the approach of a storm, and which are generally ascribed to the *moisture* in the atmosphere.

A study of the mechanism, by which the congestions observed in the caisson disease are produced, will serve to illustrate the action upon the system of the comparatively trivial changes of the barometer, just as the effect of drugs, taken in excessive or poisonous quantities, may throw light upon their action in medicinal doses.

It is obvious, that, if the blood were exposed to an equal pressure in all parts of the body, there would be no change in its distribution. It is equally clear, that the blood, if free to move, will pass from a place where the pressure is greater to one where it is less. The body is made up of structures of different densities, which present a varying resistance to compression. But, permeating these structures in every direction, are vessels in perfect communication throughout the entire system, and filled with a mobile fluid which is free to change its locality in obedience to any force which is brought to act upon it. Now, when the surface of the body is subjected to an even pressure on all sides, the tendency is to a distribution of this pressure toward the centre. If the body were composed entirely of solids, this could be effected only by the compression of those solids, and a point would very soon be reached, where the resistance would balance the compressing force, and the parts lying more toward the centre would remain unaffected. But the presence of a fluid in the structures, with free channels in which to move, changes all this. While the solid tissue resists compression, the fluid blood retreats from the surface to the centre, and accumulates there, until an equilibrium of pressure is produced.

Hence, we deduce the law, that under high atmospheric pressure, the centres will be congested at the expense of the periphery.

But, aside from location, vessels coursing through dense and resisting organs, will be less exposed to external pressure than those passing through soft and yielding structures. Hence, a second law, that firm and compact structures will be congested at the expense of those more compressible.

But there are structures, very soft and yielding in themselves, yet enveloped in a rigid casing of bone which entirely shuts off the influence of external pressure. Hence, the establishment of the equilibrium in them is wholly dependent upon an afflux of blood. This gives us the third law, that structures within closed bony cavities are congested at the expense of all others.

In accordance with these laws, we shall find, that, while in the caisson, the condition of the different parts in regard to the supply of blood will be as follows:

The skin and the superficial structures will be anæmic.[1] The central portion of the limbs and the interior organs of the body will be congested. The solid viscera of the abdomen will be especially engorged, on account of both situation and structure. The brain and spinal cord and the interior of the shaft of the long bones, will be congested to a high degree from the operation of the third law.

These changes are not perfected until a considerable time has been passed in the compressed air. The circulation, up to this point, goes on everywhere with vigor, the change being in the relative calibre of the vessels, not in their tension. The counter-pressure becomes uniform throughout the whole vascular system, but this counter-pressure supersedes the natural muscular resistance or *tone* of the vessels, which have become passive tubes. The blood is distributed, not in accordance with the physiological demands of the different parts, but in obedience to overpowering physical force.

[1] This is shown by the pallor which is very characteristic.

This is the condition of the circulation at the moment that the process of locking out begins. Yet the changes which have taken place up to this point are not the cause of the morbid phenomena which constitute the caisson disease, else the attack would take place while *in* the compressed air, instead of after leaving it. It is evident that the *removal of the pressure*, and not the pressure itself, is the immediate cause of the seizure.

This removal is effected in the few minutes which are occupied in locking out.[1] But it is not to be supposed that the vessels will instantly assume their normal condition. They are in a state of relaxation, not only in the congested, but also in the anæmic parts; in the former, because of over-distension; in the latter, because the muscular coat cannot at once recover from its inaction. The aggregate capacity of the vascular system will, therefore, be in excess, compared to the volume of blood to be conveyed; or, in other words, there will be a lowering of vascular tension.

Hence, the circulation will be languid, and the congested parts will not readily empty themselves of the excess of blood which they contain. Especially will this be the case in the brain and spinal cord, where the conditions are most favorable for the production of congestion. The capillaries being clogged with effete blood, the nutrition of the part must suffer, and disturbance of function will result.

It is to this, I think, that the delirium and the transient loss of consciousness, which occasionally occur, are to be attributed. When the spinal cord is the seat of this condition, pain in the parts deriving their nerves from that section of the cord may result, or paralysis, more or less complete, may follow.

This appears to me to account for the phenomena in those cases, in which the local symptom is paralysis, or pain

[1] *I. e.*, passing from the caisson into the open air, through the air-lock.

of a transient or shifting character. These cases may, I think, be considered as entirely spinal in their origin. But, in many cases, there are evident local changes, such as tumefaction, rise of temperature, etc., which indicate local irritation, and which are probably due to obstruction of the vessels of the part as a sequel to the local congestion. This explanation is applicable also to those cases, in which the pain is fixed in one locality, which may be very much circumscribed, and where it persists for days without intermission, feeling, as the patient expresses it, "as if it were in the bone," where it very likely is. Such a pain presents a marked contrast to those shifting pains which have been described, and, if considered of spinal origin, would indicate a serious lesion confined to a minute portion of the cord. That such a circumscribed lesion might occur as a very rare exception, must be admitted; but that it should be present in a considerable proportion of cases, is, in the last degree, improbable.

The testimony of all observers is, that the liability to attack is directly as the duration of the stay in the caisson. This admits of an easy explanation on the theory which I have advanced. The more thoroughly the system has become adapted to the change in the circulation, the less readily it will resume its normal condition, when the pressure is removed. The congested vessels, especially, will lose their contractility in proportion to the time their muscular fibres have been upon the stretch.

Now it is evident that the changes in the circulation which take place in the caisson must occur, to some extent, whenever there is a rise of the barometer, and, conversely, that a fall of the mercury must result in changes similar in kind, however slight in degree, to those attending a change from the caisson into the open air. But it may be argued, that the phenomena of the caisson disease

require a change of pressure so enormously disproportioned to any changes occurring from natural causes, that it would, be absurd to reason from one to the other. But the fact is, that observation of the effects of high pressure reveals a difference in the susceptibility of different persons to its influence which would not, *a priori*, have been expected, and which becomes a prime factor in calculating the effect of minor degrees of condensation of the atmosphere.

Of the men employed under my observation in the caissons of the East River Bridge, a large proportion bore the excessive pressure (reaching at last to 36 lbs. additional to the square inch) without the slightest ill effect; while, on the other hand, some quite severe cases resulted from a very short exposure to the slight pressure employed in the early part of the work. For instance, a student of engineering visited the Brooklyn caisson, where the pressure did not exceed 15 lbs., and, after a very brief stay, was seized, on coming into the open air, with temporary paralysis. That a short exposure to a pressure of 15 lbs. should paralyze one man, while another was able to bear, day after day, without inconvenience, a pressure of 36, or even, as at St. Louis, of 50 lbs., is to be accounted for only by assuming a vast difference in susceptibility, the limits of which difference in either direction can only be surmised. Back of this there is probably a difference in the efficiency of the vaso-motor system, or, perhaps, in the structure of the vessels themselves, so that, in one case, the vessels resume, at once, their normal condition, when the pressure is removed, while in the other, the abnormal distribution of the blood persists in certain localities.

Whatever the predisposing condition may be in this latter class of cases, we have only to assume its existence in an exaggerated degree, to bring the subject within the range of the influence of ordinary barometric changes. If

one man can bear a change of 90 inches without feeling it, while another is paralyzed by a change of 30 inches, it is not incredible, that a third may have aching limbs as the result of a fall of 2 inches.

In point of fact we know, that there are many persons who can foretell, by their sensations, the approach of a storm, and who are in the habit of saying, "We shall have rain to-morrow; I feel the dampness in my bones." Now the proof, that the moisture in the atmosphere is not the cause of their suffering is found in the fact, that a sudden shower may saturate the earth and fill the air with dampness, without causing them to complain, nor do they feel any ill effect from exposure to the falling dew. But, whenever the glass goes down, though the air may not be sensibly damp, they experience more or less discomfort. In such persons the action, as before suggested, is probably similar to that observed in a greatly intensified degree in the caisson disease. The change from a higher to a lower degree of atmospheric pressure disturbs the circulation in a way to affect certain nerve cells or nerve fibres; the individual having a strong natural or acquired predisposition, a "neuralgic habit," which needs but the slightest cause to develop a greatly disproportioned effect.

This predisposition, or habit, may consist simply in the existence along a nerve, or at its origin, of a point, at which the capillaries are, for some reason, more than usually distensible. Such a condition of the cutaneous capillaries is seen sometimes in children who have a mother's mark that has so faded as to be imperceptible, except when the child cries, when it becomes plainly visible. Some cicatrices, also, present the same conditions, a key, perhaps, to the neuralgias following gunshot wounds, which are especially prone to be affected by changes of weather.

A curious fact, however, in regard to the terrific pains of

the caisson disease is, that they are often not felt, until several hours after coming into the open air. In these cases, it is probable, that the area of capillary obstruction does not, at first, include a centre of pain, but that it widens, as all capillary disturbance is disposed to do, until such a centre is reached.

This delay in the development of neuralgia from lessening of pressure, would tend to obscure the study of cases in connection with barometric changes,[1] the pain, perhaps, coinciding in point of time with a rise, though caused in reality by a fall, of the barometer.

No one can have failed to remark the difference which we feel in our mental and bodily efficiency in different states of the weather. On clear, bright days the brain is active, the muscles vigorous, and the internal organs appear to work smoothly. On damp and foggy days, on the contrary, mental effort is irksome, the limbs drag, the appetite is less, the digestion slower, and the whole tone of the system is lowered.

This difference may be explained, at least in part, on the principle under discussion. When the air is clear, the barometer is usually high, and the greater pressure upon the surface drives the blood to the interior of the body, and especially to the organs in closed cavities—such as the brain, and to solid and dense organs—such as the liver and kidneys, thus stimulating their functions. At the same time, the pressure assists the muscular tone of the vessels in diminishing the total vascular area, and thus insuring celerity of the blood current everywhere. But when the pressure falls, as it does in damp weather, the peripheral vessels, deprived of a part of their support, yield to distention, and there is a transfer of blood to them from the more central organs, and, at the same time, a general slowing of the circulation, all resulting in lessened vital energy.

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[1] For a very interesting study of this kind, see a case reported by Dr. Weir Mitchell, *Am. Jour. of Med. Sci.*, Jan., 1877.

How much these changes have to do with initiating disease, is a question which opens a wide field for conjecture. That increased pressure on the surface might be sufficient to determine the rupture of a miliary aneurism in the brain, is easily conceivable. That diminished pressure might concur with other causes in bringing about internal congestions and inflammations, is at least probable; and that the lowered vital tone from a sudden fall of the barometer may render the system an easier prey to other causes of disease, is a justifiable inference.

There are many forms of disease that recur at irregular periods in persons susceptible to them, the subject being, in the intervals, apparently in perfect health. There are, evidently, in these cases a predisposing cause, which is permanent, and an exciting cause, which is transient. The former is inherent in the individual; the latter is some influence operating from without. Neither is capable alone of producing an attack, their joint action being required.

The predisposing cause, for the most part, eludes our observation. The exciting cause is often sufficiently apparent and may be error in diet, exposure to cold, over-fatigue, mental excitement, etc. But sometimes the strictest inquiry fails to elicit the cause, although some must have existed. That some, at least, of these cases are attributable to such disturbances of the circulation as have been mentioned as depending upon change of atmospheric pressure, appears to me to be more than likely.

At this time, when the state of the barometer at any given hour is a matter of permanent record, accessible to all, it would not be difficult, especially in hospitals, to compare notes of cases with barometric tables, and results of great scientific value might be obtained.

A STUDY OF THE PHYSIOLOGICAL AND TOXIC EFFECTS OF GLYCERINE IN LOWER ANIMALS.

By R. W. AMIDON, M. D.,

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WHILE studying the effects of arnicine in the frog during the last summer, at the Physiological Laboratory of Dr. Ott, at Easton, Pa., it was noticed that no preparation of the drug had much effect except a solution in glycerine. This solution produced such striking and constant symptoms that a suspicion was aroused that they were due to the glycerine and not to the arnicine, and a few experiments confirmed this view. The fact that glycerine so often enters into solutions of drugs physiologically studied, and that it might in this way mislead the experimenter, led to a detailed study of its effects on frogs and rabbits, the results of which are given herewith. Many of the experiments were conducted by Dr. Ott himself.

The general effect in frogs is as follows: If from three to ten drops of glycerine are given hypodermically, pure or diluted, to a frog, the first effect is, of course, the ordinary contortion elicited by the local action of the drug on the sensory nerves.

In from five to fifteen minutes, however, it will be noticed that the frog, if induced to jump, will sprawl a good deal,

and seems to have rather hard work to retract his hind legs. This, it will be seen, is due not to a paresis of the legs, but to a stiff condition of the same From this time forward very frequent fibrillary contractions will be noticed in all the voluntary muscles. Soon, if the animal be carefully examined, it will be noted that the lightest pressure of a muscle will throw it into a momentary tetanic spasm, while irritation of a purely sensory part, as the toe, will cause attempts at ordinary reflex action. Generally in the course of one half an hour, or sooner if the dose be large, the frog will be thrown into a general tetanus, the fore legs adducted and crossed over the chest, the hind legs rigidly extended. Now the muscular hyper-excitability will be very marked. Light pressure will throw the irritated muscle into a violent tetanic spasm, so that the limbs may be made to assume temporarily any attitude desired, the original attitude being assumed after the irritation is stopped.

Painful impressions on a sensory part will cause an evident endeavor on the part of the animal to shrink and draw himself away, a thing which is of course impossible on account of his extreme rigidity. There are indubitable signs of pain, however. The tetanic state invades all voluntary muscles, respiration ceases, while the circulation continues for some time, the heart finally stopping in an apparent diastole; the auricle fluttering for a long time after ventricular contractions cease. The tetanic condition does not relax with death, but passes uninterruptedly into an extreme rigor mortis.

Given in larger doses by a vein or under the skin of a rabbit, a somewhat similar but not identical train of symptoms follows.

Two or three hours after the administration of from sixteen to thirty cubic centimetres under the skin, and sooner after a larger dose, a rabbit begins to appear weak, his legs

slide out from under him, and he sinks down apparently from sheer exhaustion.

The heart becomes weak and irregular, the respiration superficial, the head becomes tremulous and droops, fibrillary twitchings are seen in the head and all four extremities, and finally a general clonic spasm runs through the animal, respiration ceases, while the heart continues to beat feebly for some minutes longer.

One to five cubic centimetres introduced into a vein cause a rather sudden stoppage of both respiration and circulation, preceded by a general shudder. The exalted muscular excitability seen in the frog is not as marked in the rabbit, and the tetanic condition is not seen. Hæmaturia follows both modes of administration of the drug provided the animal does not die suddenly.

Experiments with the mercurial kymograph of Ludwig showed that, in the rabbit, the intravenous injection of one to two cubic centimetres of glycerine caused immediately a rise, followed by a fall of blood pressure, accompanied by diminished force, increased frequency, and irregularity of the cardiac contractions.

If the dose given were small, the circulation would seem

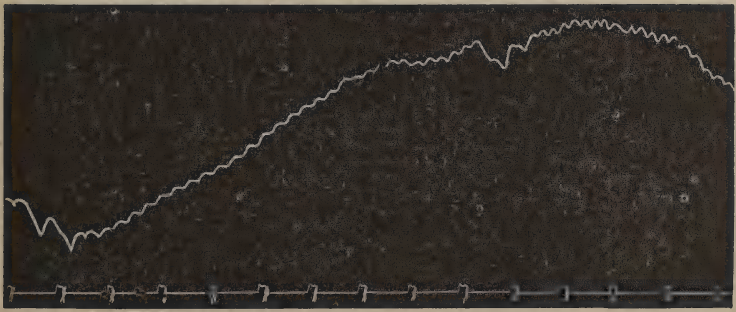

FIG. I.

Curve showing immediate rise in blood pressure.

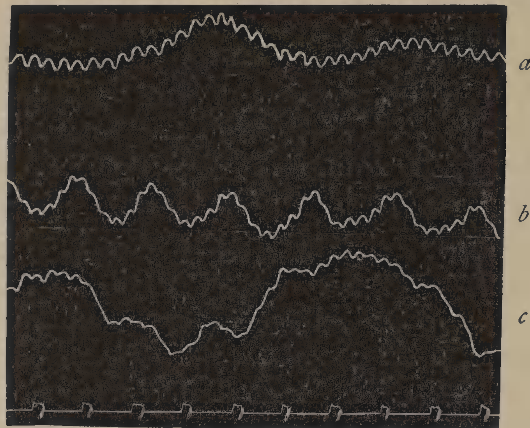

FIG. 2.

Curves showing changes in blood pressure.

	PULSE.	PRESSURE.
a. Immediately before first experiment	210	104 mm.
b. One minute after injection of glycerine . . .	264	92 "
c. Two minutes after injection	222	90 "

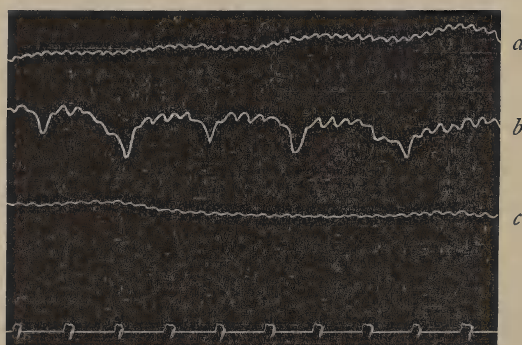

FIG. 3.

Curves showing changes in blood pressure.

	PULSE.	PRESSURE.
Before second experiment		104 mm.
a. One minute after glycerine injection	282	94 "
b. Two minutes after	204	96 "
c. Four minutes after a second injection given at the seventeenth minute	246	84 "

to recover a perfect equilibrium in a short time (quarter of an hour to one hour), while if a large dose (two cubic

centimetres) were given, the blood pressure would rapidly fall, the pulse become rapid, feeble, and finally extinguished, and the animal would die, seemingly of cardiac paralysis.

To demonstrate that the effect of the glycerine was peripheral and not central, an experiment was performed, the results of which are given below, and which show similar but modified results to former ones. The medulla oblongata and the cardiac nerves on both sides of the neck of a large rabbit were exposed, and when the connection with the kymograph was established as usual, both the medulla and the cervical cardiac nerves were cut.

TIME. SECONDS.	PULSE. RATE PER MINUTE.	PRESSURE. MM. OF MERCURY.
1st	240	32
15th	220	32
	injected 1.3 glycerine.	
39th	240	32
40th	indistinguishable	34
41st	"	32
42d	"	30
43d	"	26
46th	"	20
47th	"	19
48th	"	18
50th	"	16
62d	"	14

To ascertain whether glycerine stops the heart by irritating the peripheral pneumogastric, or by direct action on the heart substance itself, the peripheral pneumogastric was paralyzed by the administration of atropia, and then glycerine was given, with about the same results as before, thus showing that very likely the circulatory changes are due to direct action of the glycerine on the heart. If now in this atropinized and glycerine-poisoned animal the sciatic nerve be severely excited by a strong faradic current, a sudden

and large rise of blood pressure will ensue, showing both that the centripetal paths are open, that the vaso-motor centre is intact, and that its centrifugal fibres conduct impressions.

The accompanying curves show the effect of glycerine on the respiratory function of the rabbit. An experiment where the vagi were previously cut showed no peculiarity over the other except that the effects of the drug were for some reason delayed. The respiration is at first made slow and more shallow, and later, before death, is distinguished by deep respiratory movements and a long respiratory pause. The gradual failure of respiration is synchronous with the failure of the heart, and is undoubtedly due to the same cause.

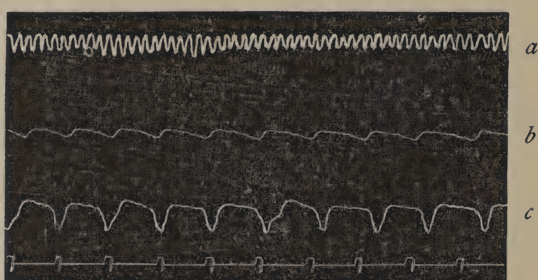

FIG. 4.

Respiratory curves in rabbit poisoned by glycerine. *a*, before injection ; *b*. one minute after ; *c*, four minutes after, just before death.

The general symptoms in the frog have been narrated. It remained to ascertain on what part of the nervo-muscular apparatus the drug exerts its power. The following is a *résumé*, in as few words as possible, of the experiments performed to arrive at a decision on that point : A frog already tetanized with glycerine will remain so if his whole central nervous system, brain, medulla, and cord, be destroyed. A frog whose brain has been cut off before the administration

of the drug will become tetanic. If the medulla be cut off the same result will follow, only more slowly, because of the sluggish circulation. Again, section of the nerves lead-

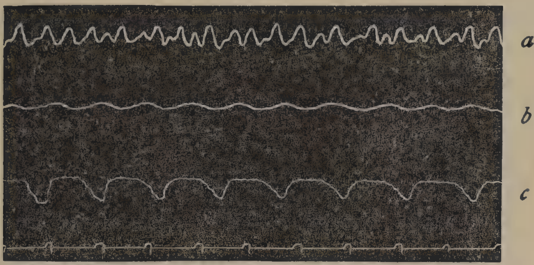

FIG. 5.

Respiratory curves in rabbit with vagi cut, and poisoned with glycerine. *a*, after section before glycerine; *b*, ten minutes after; *c*, twenty minutes after, just before death.

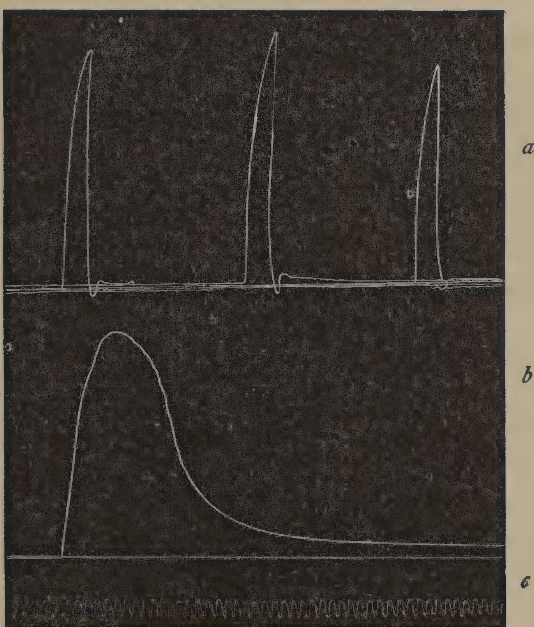

FIG. 6.

Traces of muscular contraction taken with Marey's myograph. *a*, the curve of normal muscle; *b*, the curve of a muscle poisoned by glycerine; *c*, trace of tuning-fork vibrating at the rate of 60 vibrations per second.

ing to the limbs has no effect on the spasm. By cutting off the blood supply to any part, either by ligating the abdomen, leaving the cord intact, or by ligating all of a limb, except the nerve, it is found that no tetanus ensues in the protected part. This narrows down the field to either the motor-nerve termini or the muscular substance itself. Glycerine given after or with curare produced the tetanic symptoms, thus showing that, without doubt, glycerine attacks the muscular fibre itself.

There are appended myographic tracings of the muscles from healthy frogs and of muscles from frogs poisoned by glycerine, the curves of which exhibit the most marked differences.

In the case of the healthy muscle it will be noticed that the contraction is of very brief duration, occupying only from one thirtieth to one twenty-fourth of a second, while in the tetanized muscle the contraction lasts from one half to three quarters of a second.

If the brain of a frog be removed, it will be found by the sulphuric acid test that glycerine diminishes the reflex irritability. If, then, the medulla be cut off, reflex action becomes exaggerated, showing that the former depression of reflex irritability is due to an excitation of the centres of Setschenow in the medulla oblongata.

It is thus seen that glycerine, particularly in the frog, has physiological effects which should lead to its abandonment as a solvent or diluent of other drugs in physiological experiments.

LACERATION OF THE CERVIX UTERI.

By H. J. GARRIGUES, M.D.,
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DR. EMMET has called special attention to a cicatricial plug often formed in the angles of a lacerated cervix, and to the importance of its removal.[1] A short time ago the doctor brought me two bodies which he had removed from such a case, digging them out like bullets embedded in flesh, and requested me to examine them, as no description of this tissue had been published hitherto.

These bodies were of irregular, roundish shape. The largest measured 11 by 9 millimetres, the smallest 9 by 7 millimetres. They were of slightly yellow-red color, translucent (they had been put in a mixtnre of glycerine, alcohol, and water), and composed of a dense elastic tissue.

The larger of them was hardened in a solution of chromic acid, and cut perpendicularly to the surface. The sections, stained with carmine showed an epithelial layer, a mucous membrane, and a cicatricial tissue.

The *epithelium* is very thick and composed of two zones. The upper zone, corresponding with the *stratum corneum* of the epidermis, is composed of large flat cells, each with a nucleus, with the exception of a few of the most superficial which have become quite flat and horny, but, as a rule, even

[1] Emmet, Principles and Practice of Gynecology, 2d edition, 1880, p. 473.

the superficial cells are rather thick and provided with a central nucleus. Seen in front view they are rhomboid or multangular, with fine indentations in the edge. Seen in side view they appear spindle-shaped, being thick in the middle and tapering toward both ends. They are arranged in such a way as to cover one another with one half of their length. I can count as much as twenty such rows of cells one below the other.

The lower zone, corresponding with the *rete Malpighii* of the epidermis, is only one fourth as thick as the horny zone, and composed of much smaller cuboidal cells, gradually changing shape so as to become like those in the upper zone. In the very deepest layer the cells are even a little columnar, *i. e.*, longer than wide.

In this epithelium are seen deep, narrow bays taken up by prolongation from the underlying layer. They extend sometimes into the horny layer, but are always covered all over with a layer of the mucous stratum.

Under the epithelium is seen a *mucous, or dermal layer* with papillae. The chief direction of the fibres of the connective tissue is parallel to the surface. The upper part contains some small round cells, which especially are found in large number in the papillæ. In the deeper parts the cells become more scarce.

This dermal layer changes imperceptibly into a *cicatricial tissue* composed of dense connective tissue, with bundles going in all directions. In this tissue are found quite a number of arteries and veins, but neither nerves nor muscle fibres.

This examination, then, proves that there really exists a "cicatricial plug," but that it is covered by the mucous membrane and epithelium of the portio vaginalis, which has grown over it from the torn edges.

I had hoped to find nerves embedded in this tissue, which

would have explained the singular nervous disturbances so commonly observed in patients suffering from laceration of the cervix. I have myself observed a very marked instance of this kind. The patient was a highly cultivated French lady, with an extremely developed nervous system. After having spent most of her life in easy circumstances, she lost her fortune and became a widow, obliged to make a living as a teacher. She would certainly not have gone through all the troubles and expenses of an operation if she could have avoided it, but she suffered so much pelvic pain that she could scarcely walk. She had constantly the harassing feeling that her nails were being torn from her fingers, and she saw herself surrounded by wild beasts with open mouths. From the very moment of the operation these latter sensations and delusions stopped, and after some months she was perfectly able to attend to her business, which entails a good deal of walking on her. It is now almost a year since I operated upon her, and she continues well.

But since the plug, as stated, did not contain any nerve twigs, the cicatrix must influence the nerves in a more indirect way, probably by the pressure on the underlying nerve-ends produced by the contracting cicatricial tissue.

I will seize this opportunity to add a few words about the *obstetric indication* for hystero-trachelorraphy, or Emmet's operation for lacerated cervix. The indications so far pointed out are all gynecological, such as leucorrhœa, neuralgia, anæmia, and the danger of carcinomatous degeneration. In watching recently a case of tedious labor, it struck me that the obstetric side of the question had been overlooked. It was a tripara. At my very first examination I found a thick, double-lacerated cervix. The pains were good, but this dense inflamed tissue opposed a great resistance to dilatation. In spite of a constant use of the hot

douche, chloral, and Barnes' dilators, it took twelve hours to get this hard, unyielding œdematous neck sufficiently opened to let the head pass, and even then it was only with the forceps that I could deliver the lady from the fearful suffering due to the pressure against an inflamed cervix. The child was yet entirely in the uterus, the anterior lip formed a finger-thick cushion, which prevented the head from passing the pubic arch, while the whole uterus was so far down that during tractions and after delivery the anterior lip was visible in the vulva.

Such cases ought to be operated on before another pregnancy supervenes. Cicatricial tissue is cut away, a new vitality is imparted, during the healing process hyperplasia is subdued, and the restoration of the normal relations of the parts allows a free circulation to go on. Thus the cervix becomes fit to play its part in the next confinement, so as not to disturb an otherwise natural labor.

A few years ago I treated a young lady who had a double laceration dating from her early confinement. Before she came under my treatment she had been treated for months and then sent to Europe on account of her constant ailing. After having replaced a retroflected uterus, and kept it in position by aid of a Peaslee's flexible pessary, and treated her leucorrhœa in vain during four months with hot water injections and astringent applications, I performed hysterotracheloraphy with perfect success.

The leucorrhœa stopped, the backache of which she had suffered for years was gone. I advised her to renew her marital relations, which had been entirely interrupted for years on account of her health. She conceived immediately, bore a child without the least trouble, nursed it herself, and was, in fact, restored to health and enjoyment of life.

These two cases, I think, illustrate the obstetric side of

the question; and show, on one hand, how a lacerated cervix may be a serious impediment to labor, and, on the other, how a cervix which has been restored by Emmet's operation, performs its function naturally during labor, and even may come through this ordeal without sustaining new injury.

THE PROPHYLAXIS OF INSANITY.*

By MARY PUTNAM JACOBI, M. D.

A TERRIFIED popular imagination still pictures insanity as some mysterious and monstrous incubus, coming from distant regions of darkness to crush out human reason. In reality, however, insanity means a complex multitude of morbid states, varying indefinitely in form and intensity, but all composed of elements which preëxist in health. This fact affords a basis for prophylaxis, for it indicates the possibility of detecting these elements and, to a certain extent, of anticipating their morbid combinations.

There are as many degrees in the soundness of men's minds as in the soundness of their digestions. Study of the organism of the family, sometimes in several generations, often serves to detect flaws in the individual organization otherwise too minute for notice. It is to the family organism that especially applies the doctrine of the blending of apparently opposite elements,—as genius and insanity, both springing from an unstable equilibrium of the nervous system. These elements sometimes, though rarely, blend in the same person. But far more frequently it is inheritance from the undeveloped side of an organization of genius which results in an organization of imbecility.

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* A portion of a paper read before the American Social Science Association, at Saratoga, Wednesday, September 7, 1881.

The original organization gives the physical substratum; upon this the succession of psychic processes, which begin with the dawn of consciousness, builds up the mental individuality. Ideas, feelings, volitions, enter liberally into the structure of the mind,—are the constituent elements of which this has been built up. Permit me to quote the description given by the celebrated Griesinger:

"Self-consciousness,—the Ego,—" he says, "is an abstraction in which are contained, closely welded together, residue of all the sensibilities, thoughts, and volitions which the individual has ever experienced.

"* * * These are gradually aggregated into complex masses of conceptions, varying in density and resistance, according to the internal cohesion of their elements. * * * The character of the individual varies with their relative predominance; their constant struggle with one another constitutes the internal conflict which is essential to normal mental existence.

"* * * The development of insane delusions follows the same laws as that of healthy ideas. New sensibilities, volitions, and conceptions present themselves to the pre-existing conception-masses, are at first repelled by these, gradually penetrate them, and if the cohesiveness of the latter be weak or weakened, assimilate to them until the Ego is transformed or completely falsified. *In this process the previous composition of the Ego is seen to be of immense importance.* A weak (loosely knit) nature will, much earlier than a strong one, be overborne by anomalous conceptions." [1]

Thus, at any given moment, the mental organism consists not only of its physical substratum, but of that *and* of the long series of psychic processes which have been built up on it. It is a fundamental law of all organized

[1] "Pathologie und Therapie der Psychischen Krankheiten," 1867.

tissues, and most conspicuously illustrated in the brain, that function not only depends upon structure, but ends by modifying it. Hence, morbid modifications of psychic processes may be initiated either in them or in the physical substratum. This is equivalent to the previous assertion that insanity may be determined either by a psychic or a somatic cause, but generally requires the concurrence of both.

In the existing professional and popular reaction against the old puerilities of the exclusively moral theory of insanity, these facts are often overlooked or misunderstood. The question of prophylaxis has become narrowed down to the question of prophylaxis in marriage. This is not only much too narrow, and the social difficulties in the way very great, but the rules for practice have been by no means worked out, and many of those which have been suggested are erroneous or superficial.

The fact that the previous constitution of the mental conception-masses modifies the process of their falsification under the influence of mental disease, should suggest an effort to so build up this constitution that it may be fitted to resist strain. For the formation of the conception-masses is far from being a spontaneous or self-directed process. No ideas can enter the forming mind except from without, from communication with its fellows, or from the transformation of sense impressions. It is therefore largely in our power to determine the nature of the ideas of any child who is *thoroughly* guarded from his cradle. Again, the will develops in the mould it makes for itself by successive volitions; these may to a considerable extent be commanded or contrived. It follows that, hand in hand with prophylactic treatment of the physical substratum of the inherited nervous organization, should go strenuous educational prophylaxis of the psychic processes.

But there is needed a far-sighted, comprehensive, minute education, which should begin with the dawn of consciousness, and extend, if possible, through life. It should have a detailed objective or reason for each step in the elementary lesions of the disease which menaces the person, or in the elementary defects of his menaced constitution.

To assert that moral prophylaxis is useless because insanity is merely a symptom of physical disease, is to contradict the facts of the double nature and double origin of the psychoses which are admitted by the best authorities. Educational prophylaxis could only be expected to contribute one factor toward the solution of the problem; but it is one, and all the more worth considering, because at present it is so generally neglected.

A more plausible objection is that the moral substratum of minds predisposed to insanity is peculiarly perverted, so that they are insusceptible of education. That it is precisely this insusceptibility which especially manifests their predisposition.

Finally, it may be alleged that the traits of character which exist in a person before an attack of insanity, can offer no guide for treatment, because in the attack these are all reversed.

This last objection is met by the answer that the prophylaxis of mental, as of somatic diseases, is to be directed, not to the symptoms of the malady, but to the constitutional defects which facilitate its invasion, and to the circumstances of the surrounding medium which become the occasioning cause. Thus, it is known that under a great weight of responsibility a cheerful-tempered, but feeble-willed person may break down into melancholia. The prophylactic training should therefore be directed, not toward making such a person cheerful, but toward inuring

him, by gradual practice, to bear responsibility. And so for other analogous cases.

The ideal prophylaxis implies that in neuropathic families the entire life of each child, its physical and moral training, and every detail of its social surroundings, should be planned with a view to avert mental disease. According to the degree of predisposition, this is liable to occur spontaneously at ordinary physiological crises, as puberty, menstruation, pregnancy, parturition, lactation, the climacteric; or only under the influence of external causes. In the latter case, the far-sighted disposition of the social medium of a predisposed person may often avert an attack of insanity by averting the cause.

It is evident that the far-sighted and self-controlled guardianship required should be entrusted to a person not sharing the family constitution; to the parent who may be exempt, or, if both are affected, to a person who is not a relative at all. For the present purpose only a word is needed in regard to the main details of physical prophylaxis.

They are: abundance of nitrogenous food; daily cold bathing; pure air; daily exercise in it, especially by means of cultivation of the ground, the cardinal employment for the body and mind of neurotics.

A fifth point of great importance is rest; equally so for an immediately threatened attack, and in the life-long management of susceptible persons. For them over-exhaustion and fatigue are always to be dreaded, and to these they are particularly prone, from the extremely deficient power of resistance of their nervous system. It is worth noticing that it is neuropathic families more than any others who are liable to neglect the foregoing precautions.

For effective moral prophylaxis, it is desirable that a certain amount of information be popularly diffused, to facili-

tate the awakening of domestic solicitude, the recognition of incipient insanity, and of the slighter but significant marks of the insane temperament. This may prove as useful as it has already done in regard to scrofula, rhachitis, tuberculosis, and other constitutional diseases.

Krafft-Ebing ranks severe and congenital hysteria with the psychic degenerations, and shows it to be the forerunner of much real insanity.[1] Knowledge of this fact might do much to check the capricious and vacillating treatment to which youthful hysterical patients are generally subjected. On the other hand, in the permanent prophylaxis for adult life, which must so largely be committed to the patient, it is extremely useful to be aware of the relative benignity of the very forms of insanity which usually excite the most alarm. Acute melancholia, mania, and primary dementia are classed with the functional disorders or psycho-neuroses, tending, under favorable circumstances, to spontaneous recovery. This knowledge might help to avert at least those distressing suicides which are committed, not from insane impulses, but under the dread of impending insanity. They are far from proving that this has already set in, for it is really not irrational to choose death in preference to permanent dementia.

The following traits are signalized as characteristic of the neuropathic constitution—constitution which affords the main physical and moral basis for the development of insanity.

In neuropathic families the children early manifest a remarkable nervous excitability, with tendency to severe neurotic disorders at physiological crises, as convulsions during dentition, neuralgias at menstruation. The establishment of menstruation is often premature, often preceded and followed by profound chloro-anæmia. The

[1] This statement is not made in regard to acquired hysteria, symptomatic of uterine or other diseases.

cerebral functions are easily disturbed, slight physical disorders being attended by somnolence, delirium, hallucinations. The nervous system seems to be everywhere hyperæsthetic. Reaction to either pleasing or displeasing impressions is excessive; there are abundant reflex neuralgias, vaso-motor irritations. Pallor, blushing, palpitations, præcordial anxiety, are caused by trifling moral excitement, or by agents lowering the tone of the vaso-motor nerves, as heat or alcohol.

The sexual instincts are precocious and often perverted. The establishment of puberty is often the sign for the development of spinal irritation, hysteria, or epilepsy.

The psychic characteristics correspond. The disposition is strikingly irritable and touchy; psychic pain arises for trifling cause; at the least occasion the most vivid emotions are excited. The subjects of this temperament alternate rapidly from one extreme to the other; their sympathies and antipathies are alike intense; their entire life is passed between periods of exaltation and depression, leaving scarcely any room for healthy indifference.

On the other hand, there is a remarkable inexcitability of ethical feeling. Vanity, egotism, and a jealous suspiciousness are common, and the temper is often violent. The mind is often obviously feeble, with few and monotonous ideas, and sluggish association of them. At other times ideas are readily excited, the imagination is active, even to the production of hallucinations; but mental activity is ineffective because of the rapidity with which it leads to exhaustion. There is no time to complete any thing before the energies flag. The will is equally deceptive in its apparent exuberance and real futility. Its capricious energy and innate weakness is a fit counterpart for the one-sided talent or even whimsical genius which often marks the intelligence.[1]

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[1] Abridged from Krafft-Ebing.

This disposition constitutes the moral substratum which, together with the physical constitution, affords the constitutional basis for psychic disease. In it two elements are conspicuous: a profound and often unconscious egotism, resulting from the predominance of the instincts over the faculties for external relations; and a constant ineffectiveness in the maintenance of these relations,—in other words, abnormal weakness of the will. These elements reappear in insane diseases. Egotism is the nucleus of the exactions of hysteria, and determines the form of all delusions, which, whether primary, or engendered from emotional insanity, invariably centre on the depression or exaltation of self. The suspiciousness and violent temper so frequent in the neuropathic, develops easily into the technical delirium of persecution or of quarrelsomeness. The psychic hyperæsthesia common to several psychoses, but typical of melancholia, depends, on the one hand, on the same primitive egotism; on the other hand, on the weakness of the will, on account of which the normal channel from feeling to action is blocked. Pent-up feeling is always hyperæsthetic; psychic pain is the correlative of external ineffectiveness, even when not directly caused by it.

Diminished interest in external relations results in psychic anæsthesia, especially in regard to moral appreciations. This anæsthesia is again the direct correlative of the excess of instinctive and personal interests, and of the weakness of the will which fails to enlarge the scope of the personality, as it is naturally destined to do.

When the will is feeble, sluggish, inert, the tendency of the mind to sink under pressure, and especially under the weight of responsibility, is very great. "The fact of human freedom," says Griesinger, "is the fact of the conflict in consciousness of opposing ideas, and of the termination of

the strife by the conception-mass representing the Ego, which assimilates part of the ideas, and represses the rest." Feeble natures cannot bear this conflict without excessive pain, to which, at last, they not unfrequently succumb. In melancholia, the consciousness of diminished will power is a prominent and most painful symptom of the morbid state.

The feebleness of the will may be manifested, not by sluggishness, but by infinite caprice and incessant vacillations. This may reflect a torrent of incoherent ideas; or it may represent so rapid a transformation of an idea into an impulse, that the latter alone seems to exist. Here the channel from the internal to the external world is not obstructed; its resistance, on the contrary, is abnormally diminished; yet the volition is still ineffective. Effective volitions demand distinct and correct ideas of the external medium upon which they are to be expended. But one of the most essential elements of insanity, and of the constitution predisposing to it, is the diminution in the number, force, variety, and accuracy of the ideas held concerning the external world, and on the relations of the individual to it. This monotony of ideas is sometimes, before the attack, concealed behind desultory verbiage. Sometimes, during the immediate prodromata of an attack, it is temporarily replaced, even in feeble-minded people, by an unwonted vivacity and power. Completed delirium, however, is always monotonous. Correlated to the egotistic instinct, it always centres on the personality of the individual, which is outrageously oppressed or illimitably exalted. The ideas are few; their asssociations sluggish; memory and attention are weakened even to extinction.

A deficient power of attention is generally a marked characteristic of the neuropathic state; it lies at the basis of the irritable impatience which is so frequent in it. This leads to the formation of loosely knit conception-masses,

ready to assimilate anomalous notions. The mind is naturally credulous; unapt for criticism. It offers less resistance than another to the invasion of false ideas.

Thus the three great elements in the moral substratum of a person predisposed to insanity, are: the egotistical predominance of the instincts over the faculties of reflection and external relation; the ineffectiveness of the will, even when this is impulsive or violent; the inaptitude for ideas, resulting in their poverty and imperfect combination. The whole nature is shrunken upon itself; there is not enough vital turgescence to expand it to its normal circumference and to the points of contact of this with the external world.

The cardinal point in the management of such natures is, therefore, the expansion of their shrunken individuality. This is to be effected by means of a strenuous educational system, directed at once toward the repression of the egotistic instincts, the enrichment and systematization of the ideas, and, through multiplication of acts and external relations, the energizing of the feeble will.

The scope of the method will be made clearer by some examples. Thus: grief is an efficient moral cause of insanity. That it does not more often render people insane, is indeed a remarkable proof of the resources of the healthy human organism. However various the occasions for grief, yet in so far as these all imply personal loss, the principle of their influence is always the same.

The mind becomes so concentrated on the thought of this loss, that the latter acquires the ascendancy of a fixed idea. Apart from physical disease, the inability of diversion is great, in proportion to the habitual poverty and monotony of ideas; to the fewness of relations with the external world; to the preponderance of habitual interest in matters relating to self: to the inertness of the will, unable by

vigorous action to expend externally irritations of psychic pain.

Similarly, when disappointment or humiliations, great or small, real or fancied, are the cause, or injuries, or the suspicion of injuries, the power of the predisposition and of the occasioning cause being constantly in inverse relation to each other, we reach a grade of exaggerated hysteria or hypochondria where the egotistic instincts become able of themselves to generate melancholy, irritability, and delusions.

In another class of causations, shock plays a prominent part. Inability to resist shock is partly proportioned to poverty of ideas, which permit overwhelming surprises; partly to habitually unrestrained emotionality; partly to the passivity which prevents quick reaction. Analogous is the effect of strain, of excessive anxiety, of long-standing care and responsibilities. Healthy and justly proportioned indifference is essential to healthy equilibrium; an excess of sensibility over reflection or will power, predisposes to insanity under sufficient irritation. All experience shows that an excess of egotistic sensibility is far more dangerous than an excess of sympathy, the latter being indeed extremely rare in the neuropathic constitution. It may become a cause in non-constitutional insanity. Another line of causation is that in the direction of ideas, where the invasion of false ideas is facilitated by habits of credulity, superficial reasoning, loosely knit conception-masses. An unreflecting enthusiasm easily embraces exciting doctrines, as in the various religious or political manias, or is carried away by suggestions which covertly appeal to the egotistic instincts, flattering or alarming them, or submits to incongruous beliefs, as in the so-called partial insanity or monomania,

Perhaps none of the details of an educational prophylaxis

are foreign to the principles theoretically advocated for ordinary education. But in this they are applied, if at all, in a manner so lukewarm and vague as would render them useless for so grave a problem as the prophylaxis of insanity. To consider these principles in the order already enumerated: the repression of egotistic instincts demands effort in two directions. Negatively, these are to be atrophied by a studied atmosphere of indifference to caprice, violent tempers, ridiculous pretensions, exorbitant exactions; none of which are allowed to be gratified. In this permanent atmosphere, created by the mind controlling and guarding the child, he may learn to appreciate his insignificance relatively to the external world. Toward this and its interests he is secretly apathetic, except so far as they may be made subservient to his own vanity. The principle of justice, based on the simple fact of primitive equalities, must be profoundly in-wrought, by practical exercises, into the consciousness of the neurotic. He is naturally inclined to submit every thing to the test of his sympathies and antipathies; and the cultivated habit of reference to simple justice instead, will save him from innumerable entanglements, perplexities, and agitations, most dangerous to his mental equilibrium.

The multiplicity of human interests, the vastness and importance of the interests of the world, as compared with his own, may be impressed upon the child's imagination in many ways, if ingenuity be not lacking. The incidents, utilized or contrived, necessarily vary with the age of the child, but the same complex end is always to be held in view: restoration of the normal proportion between egotistic instincts and faculties of relation, and excitation of healthful ideas through healthful practical experiences and association with the fortunes of his fellows. Sometimes, together with mental vivacity, sometimes with mental in-

ertness, the mind of the neuropathic individual is apt to be really indifferent to intellectual relations, to knowledge for its own sake, to disinterested curiosity, the happiest appanage of a sound intelligence. Interested motives must be skilfully supplied, sufficiently to provide for the acquisition of knowledge essential to the enrichment of ideas, yet with caution, lest vanity and *amour propre* be unduly stimulated.

The acquisition of knowledge, the training in morals, the formation of habits of thought, must all be centred upon practical activities. It is the proper development of these which is to be relied upon to energize the feeble will; to accustom it to effectiveness by training to productive industry; to broaden and deepen the channels from internal concepts to impulses; to provide thus for the overflow of dangerous irritations; to check the flightiness, frequent forerunner of insane impulse; to widen the range of interests and of correlative ideas, and hence of resource against shock, vexation, and misfortune; to moderate inordinate vanity by submitting its pretensions to practical tests; to regulate moods by habits of daily labor; and to enlarge the entire personality, for the future as well as the present, by insuring, from internal pressure, the creation of a permanent career. This latter element of prophylaxis might well save from insanity many of the "lazy and languishing young ladies" whom Mortimer Granville complains of as filling private insane asylums.

It is not enough to attempt to widen the range of ideas. In some directions, and unguarded, this proves simply disastrous to persons of innately feeble intelligence. They must be trained in the formation of practical concepts; associated as much as possible with practical facts, with sense impressions, and with experiences in action. Clearness, definiteness of ideas, their frequent association with images, af-

ford no inconsiderable safeguard against morbid mental confusion. Similarly the careful training of the senses in various techniques contributes much toward the steady outward direction of nervous energies, which is needed to counteract the tendencies to internal concentration.

In this connection gymnastic training has a mental as well as a physical influence. It would be difficult to prove that such training of the periphery of the nervous system could counteract the development of hallucinations, which are caused by central irritation of the sensory centres. But it certainly lies in the line of such counteraction.

If it be important to fill the mind with concrete ideas, it is at least as important that these be correct, and not liable to be uprooted in later life. This liability constitutes a real danger in the notions of popular theology, which are so loosely allowed to be acquired even by guardians who do not believe in them. To persons predisposed to insanity, the uprooting of fundamental ideas can by no means be performed with impunity. It is important to train such persons early in a sound and simple philosophy, which shall provide a firm basis for thought and life without inviting to speculative thinking.

Finally, since the object to be gained is firmness and strength for the mind in dealing with its own concepts, practical exercises in the elementary intellectual acts are extremely important. These are but feebly carried out in ordinary schools, because the object in view is not distinctly perceived or firmly grasped. The first signs of failing mental power are, loss of memory, of power of association of ideas, of summoning contrasting ideas into consciousness, of reproducing or comparing or criticising them. It is indicated, therefore, to train the mind in advance to profound habituation with these various processes. Such training will avail nothing when physical lesions have begun to de-

stroy the intellectual mechanisms. But it may avail much in the cases where the integrity of these first becomes impaired from obstruction of function and psychic disability.

One other detail deserves notice, for it rarely receives attention. In minds predisposed to insanity there is often, perhaps always, a marked deficiency of elasticity. An impression sinks and remains; the mind cannot disengage itself nor recover its tone; it cannot pass quickly enough into the contrasting mood: a capacity to do this is the natural provision against strain: it probably corresponds to a law of rhythmic action in the physical mechanisms of thought. This capacity should, therefore, be carefully cultivated by encouraging alternations of attention at the first sign of fatigue. The contrary practice of forcing an immature mind to continued attention while under the influence of fatigue, instead of teaching it how to quickly change, is the habit of common-place education. Injurious to all, it is especially so to persons predisposed to depressing forms of insanity. It exhausts still further the elasticity in which they are naturally deficient.

The management of the perverted instincts of neuropathic constitutions may, when these are advanced in deterioration, prove a hopeless task. At a less severe degree, however, many bad propensities may be held in check by a skilful combination of the methods of punishment,—emulation and distracted attention.

One difficulty in guiding these cases generally lies in the fact that their pathological nature is not early recognized. Children are incessantly moralized, whose minds do not contain any conceptions of morals, and only an imperfect mechanism for ethical functions. According to the degree of imperfection, such persons must be dealt with as animals, who can certainly be trained into habitual lines of conduct, even though destitute of the corresponding abstract ideas.

One morbid appetite calls for special mention, that, namely, for alcoholic liquors. This, like the others, is often manifested early in life, and, as known, is not only a symptom of a neuropathic constitution, but, when indulged, a potent occasional cause of insanity. The management of this appetite is a most difficult problem. It has been plausibly suggested that the permanent and moderate administration of alcohol in the form of beer, might, with other treatment, help to avert the development of the irresistible craving.

Such are the abstract principles of a system of treatment which, if seriously carried out, properly associated with physical treatment, and so arranged that every other consideration should be subordinated to the attainment of its ends, should prove of real value in helping to avert many cases of insanity.

THE INFLAMMATORY ORIGIN OF TUMORS.*

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THE study of the cause of tumors has been one of the deepest interest to physicians since the earliest days in medicine; chiefly because an unfathomable mystery surrounds their origin. For several years I have been attracted to the study of tumors, and after careful consideration of clinical facts, and close histological studies, I have come to the conclusion, that "mystery" or "obscurity" should not be associated with their etiology. In this paper I shall endeavor to prove my proposition, viz.: that nearly *all tumors are direct products of inflammation.*

The idea of regarding tumors as inflammatory products is not new. John Burns[1] described medullary cancer as a sponge-like inflammation. The French school in the early part of this century, particularly Broussais[2] (1826), regarded all tumors as products of chronic inflammation. More than this, even Galen[3] in the second century, 1700 years ago, says that "nasal polyps are due either to inflammation or develop from a node (Phyma) or from germinal matter (Blastema)." Virchow says that many tumors are undoubtedly the products of inflammation, and that it is dif-

* Read before the Pathological Society of Philadelphia, April 28, 1881.

[1] Dissertation on Inflammation, Glasgow, 1800.

[2] Broussais, Histoire des Phlegmasies Chroniques, Paris, 1826.

[3] De tumoribus praeter naturam. Cap. 17, quoted after Virchow, Die Kr. Geschwülste, i, p. 35.

ficult to draw a line separating them from those tumors whose origin cannot be ascribed to inflammation.

In the etiology of tumors there are, after Virchow,[1] to be considered three causal conditions: first, local causes upon which depend the development of a tumor in a particular place; second, a general predisposition; third, a general cause, which, for the sake of convenience, is made to relate to the fluids of the body and is called dyscrasia or cachexia. "Cachexia," Virchow says, "is not always present. It never has been observed in the beginning of the formation of the tumor, but always as subsequent to it, and the condition can be brought about by other than cancerous juices. Cachexia will manifest itself only in such persons in whom the stomach, liver, or lymphatic apparatus becomes primarily or secondarily affected by cancer or any thing else. There is a physiological predisposition in certain tissues to be more readily attacked and sooner affected by disease than others. Highly organized tissues are very little predisposed to excessive abnormal formative activity. The latter occurs more in the connective tissue and its derivatives and the lymphoid and epithelial tissue; in the epithelial tissues, the new-formation takes its departure usually only from the younger undeveloped cells (*i. e.* from the lower stratum of the *rete mucosum*). This predisposition in tissues may be acquired; it does not need to be inherited.

"We also know that on mucous surfaces tumors occur for the most part in such places where there previously was a simple inflammatory disturbance; *e. g.*, where the simple inflammatory hyperplasia of chronic catarrh precedes the growth of polyps, and polyps may develop into cancer. In cancer of the stomach there is always seen a gradual transition of the catarrhal products to carcinomatous structure. Exostoses, warts, elephantiasis, are all preceded by inflam-

[1] Die Krankhafte Geschwülste, i.

mation. We next come to certain regions of the body which, from their situation and the character of their function, are exposed to irritation and injuries. This renders them especially predisposed to take on a diseased nature: *e. g.*, the stomach, sexual organs, bones, skin, and the different orifices. If we make an estimate of all malignant growths we find that most occur at orifices and at the orifices which are exposed to the greatest injury, consequently those of the digestive and sexual apparatus are most affected."

Virchow properly remarks: "If there would exist primarily a specific dyscrasia we would not have a single primary tumor focus from which metastasis proceeds, but we would have tumor eruption in all possible places without any definite plan."

All these facts together lead Virchow to the conclusion that even in malignant growths the primary growth does not come from a dyscrasia, and that every tumor is local and frequently of inflammatory origin.

These are the doctrines for which Virchow alone receives credit: but if we look through the older literature and even through the native literature of America we shall find a book written twenty years before Virchow's time, in which those fundamental ideas concerning tumors are set forth in all their essential points. I have reference to Prof. S. D. Gross' work: *The Elements of Pathological Anatomy*, published in 1839. I shall quote only a few sentences. "Tubercle," Prof. Gross says, "is always the result of inflammation, and that this is the case likewise with scirrhus, seems sufficiently evident from what has been stated in regard to its exciting causes. Very frequently, it is true, the disease arises imperceptibly without local injury or obvious constitutional derangement. But this certainly does not prove that inflammation is not concerned in its production. How

often do we find traces of inflammation after death, without having had the slightest indication of it during life. The fact, then, that inflammation is not manifested always by the usual phenomena, does not invalidate the idea of its presence." In another place he says: "Predisposition must also be accounted for and, in some instances, it seems to be connected with a hereditary taint, being transmitted from parents to their offspring." Hence he believed in the local inflammatory origin of tumors and conditions which predispose to them.

Dr. Woodward, U. S. Army, in his excellent paper on the structure of cancerous tumors,[1] agrees also with Virchow, saying: "The origin of the first growth was always to be looked for in local influences. Former injuries of one kind or another could be affirmed in a large number of cases to have preceded the development of the disease, and though frequently the patients had lost all recollection of the original harm, yet it was in this direction we ought to look, rather than seek to explain away the real difficulty by invoking the aid of an imaginary cachexia."

Although Dr. Woodward at another page says: "The time has not come yet for any one to tell why cancer originates," he must be classed with the authors of the inflammatory theory of tumor formation, as he expresses the view that cancer cylinders are largely formed from migrated white blood corpuscles; the latter when infiltrating tissues being always of inflammatory origin.

Samuel, of Königsberg,[2] although agreeing with Virchow in all the essential points, maintains more strongly than anybody else the view that inflammation is the main cause of tumors. He says: "The idea of excluding or even limiting the causative relation of chronic inflammation to neoplasms, leads far astray from the right path."

[1] Toner Lectures, Washington, 1873. [2] Handbuch d. allg. Path., 1879.

The main reason for this view of Samuel is obvious. Like Virchow he cannot conceive of the difference between permanent inflammatory products and tumors, and he classes the products of specific inflammation, *e.g.*, gumma, tubercle, lupus, etc., with the tumors.

Before giving my own views, I like to mention briefly the other theories on the etiology of tumors.[1]

Cohnheim, of Leipzig,[2] holds rather an exclusive view. Regarding tumors as typical new-formations of embryonal origin, he classes them as malformations, forming a subdivision of "monstra per excessum." True tumors, according to him, cannot originate by virtue of any kind of interference; only one causal factor exists, viz., an "*anomalous embryonic arrangement*."

Similar views had been so far expressed by other pathologists in regard to the origin of the dermoid tumors alone. These have been proven by Lücke and others to be due to anomalous invaginations of the outer layer of the blastoderm during the formation of the structure of the eye, mouth, neck, the ovaries, the testicles, etc.

Cohnheim applies to all neoplasms a similar mode of origin. He explains the anomalous embryonic arrangement which forms the starting-point and becomes the cause of tumors, by the following hypothesis: "In an early stage of embryonal development there may be undoubtedly produced more cells than are necessary for the construction of a certain part; so that a certain number of cells remain superfluous. Their number may be very small, but they possess great proliferating power on account of their embryonal nature. This must occur before the complete differentiation of the blastodermic layer and the formation of

[1] For the details of these views, and a complete record of experiments made in connection with tumors, see my paper, "The Etiology of Tumors," 1881, which can be obtained from the Secretary of the Pathological Society of Philadelphia, or by writing to my direction.

[2] Allgemeine Pathologie, 1877.

organs." This appears to him the easiest explanation, why, from such a misplacement, there occurs, not the undergrowth of a certain part of the body, *e.g.*, giant leg, but simply a histoid tumor; *i.e.*, it results in an excessive undergrowth of only one tissue of the part. The superfluous cell material may be distributed uniformly, or it may remain together in one place. In the first case there will result a superfluous part of the system, like supernumerary fingers; in the second case, a tumor.

Germs may fail to develop on account of the lack of necessary stimulus, or because of the resistance of surrounding structures.

Epstein[1] is a decided believer in Cohnheim's embryonal hypothesis. Having discovered epithelial pearls in the mucous membrane of the gums, the tongue, and the genitals of new-born infants, he thinks to have found a proof for Cohnheim's view, regarding these pearls as the famous supernumerary embryonic collections of cells. These having no physiological purpose may either disappear, or under certain conditions of nutrition form the starting-point for tumors.

Prof. Maas, of Freiburg,[2] declares himself strongly in favor of Cohnheim's theory on the etiology of tumors. He rejects positively all other theories, admits, however, traumatic influences in a greater extent than Cohnheim does. He even thinks that an injury frequently induces the development of tumors, but only in such parts where supernumerary embryonic cells exist. He says that only in such places containing those supernumerary cells an injury will react, starting a tumor by a "tumor-producing proliferation of cells"; while an injury inflicted upon a part where the cells are normal, will never produce that effect. By this

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[1] Epstein, Ueber Epithelialperlen, etc., *Zeitsch. für Heilkunde*, i, 1880.

[2] Maas, Zur Etiologie der Geschwülsten, *Berliner Klin. Wochenschr.*, No. 47, 1880.

hypothesis he tries to explain why thousands of injuries are not followed by the formation of tumors, while in a few instances tumors develop promptly in consequence of a trauma, and this only because in these few instances there must have been present abnormal embryonic cells which gave rise to the development of the tumor.

Hence he thinks that even a traumatic theory of the development of tumors can be established only by Cohnheim's hypothesis.

He quotes several cases where nævi and other congenital formations developed into cancers in consequence of an injury; the latter, however, not being necessary in the presence of the numerous cases of congenital growths on record, and the large quantity of cases of tumors in which a traumatic history cannot be traced.

Cohnheim's hypothesis on the etiology of tumors seems of late to gain some more ground in Germany. While my paper was in press, the just-issued (August 8, 1881) part of vol. 85 of *Virchow's Archiv* reached my hands, which contains an article (Exper. Untersuch ueber die Ætiologie der Geschwülsten) by Dr. Leopold, who violently supports, and thinks to have proven by experiments, the view of Cohnheim. I can give here only the mere outlines of the contents of the paper.

The author had made a series of transplantation experiments in Pathological Institute of Leipzig, under the direct supervision of Cohnheim. Essentially these experiments went only to confirm the experiments of Zahn,[1] *e. g.*, that only embryonal (fœtal) tissues transplanted into the anterior chamber of the eye or into the peritoneal cavity will grow; while adult tissues do not grow but become absorbed sooner or later. Successful results Leopold obtained only with embryonal cartilage, transplanting small fragments into the anterior chamber of eye, where they continued to grow and in about six months reached a bulk three hundred times larger than the original pieces inserted. Larger pieces of tissues and organs, *i. e.*, a whole fœtal head or a thigh inserted into the peritoneal cavity, did, however, not grow even after the lapse of

[1] Zahn, Sur le sort des tissus implantés, etc., Congrés Méd. International, 1877, Genève, 1878.

months, but were peculiarly preserved without decomposing, becoming enveloped into a connective-tissue capsule. The fragments of cartilage referred to above had not only considerably increased in size but showed advancing development, viz. : the formation of marrow-cavities and of bone lamella.

From this the author concludes, that he has produced experimentally a tumor, an artificial enchondroma (?). He thinks that he will also be able "to produce artificially in the same manner epithelioma, myoma, adenoma, and dermoids."

I think, however, that he must succeed better with them than with his "artificial enchondroma," which is nothing else than a simple graft of cartilage transforming into bone.

I must confess that Cohnheim's embryonal hypothesis is very seducing, but still it can hold good only for the congenital tumors, viz. : Rhabdo-myoma, simple angioma, and lymphangioma, dermoids, and perhaps the heterotopic adenomata. For all the rest of the tumors Cohnheim's theory is untenable, as will be shown later.

Rindfleisch,[1] in his famous text-book on pathological anatomy, expressed himself decidedly in favor of a *spontaneous origin of tumors.* His classical phrase : "Tumors arise spontaneously, but they do not heal spontaneously; while inflammations do not arise spontaneously, but they heal spontaneously"—expresses really very perfectly the notion of the practical physician. The persistence, the "organ-like character of tumors," he explains by the fact that tumors follow more the rules of physiological growths. Inflammatory formations on the other hand are produced essentially by a conflux of mobile cells at the spot of irritation, hence their rapid appearance and almost traceless disappearance.

Rindfleisch considers the evolution and involution of tissues and organs to be an important factor in the etiology of tumors. Thus, he explains the development of tumors of the connective-tissue group by a localized, excessive proliferation of connective-tissue elements during

[1] Rindfleisch, Lehrbuch der pathol. Gewebelehre, 1875.

evolution (in young persons), and the occurrence of epithelial new growths during involution (in older persons), by a local proliferation of the superabundant epithelial elements. He admits, though, that local irritation plays an important rôle.

In connection with the idiopathic or spontaneous theory due notice must be given to the views of Mr. Payne,[1] who applies Spencer's dynamical laws to the causation, or rather to the growth of tumors.

Herbert Spencer says: "Growth is unlimited or has a definite limit according as the surplus of nutrition over expenditure does or does not progressively decrease. Tumors, having no function, have no expenditure, and hence all the force is used up in growths, and the larger the tumor the more force is liberated and the larger it grows. They are like plants in being almost wholly accumulators; they have no expenditure of force, hence their unrestrained increase in size."

Mr. Payne properly remarks: "New growths are more frequent in passive tissues than in actively fluctuating tissues. Fatty tissues, bone tissues, and all varieties of growths which originate from connective tissues, are instances of the connection of mechanical passivity with excessive growth." On the other hand, he explains the extraordinary rarity of tumors composed of striated muscular tissues by the strong activity of the latter, the nutritive supply being balanced by the expenditure of force.

A *nervous theory* of the etiology of tumors is also more or less ably advocated.

Dr. Snow,[2] of London, in his paper on the etiology of cancer, based upon two years' statistics from the cancer hospital, comes to the conclusion that nervous depression, especially mental trouble, is the most prominent cause of

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[1] Payne, Origin and Relation of New Growths, *Brit. Med. Journal*, 1874.

[2] Snow, *London Lancet*, December, 1880.

cancers. After careful inquiry into the history of a large number of cases, he comes to the following conclusions:

1. "Hereditary tendency, as a predisposing cause of cancer (at all events of mammary and uterine), is almost valueless, if not entirely so, and in practical diagnosis should altogether be ignored as misleading.

2. "Mechanical injuries directly produce cancer in a certain percentage of cases, but this percentage is small.

3. "As direct and immediate causes of cancer (especially in my own experience of uterine cancer) mental trouble, hard work, are very potent agents, and exert more influence than any other antecedent within our present knowledge."

The immediate cause of tumors has been repeatedly traced, beyond doubt, directly to inflammatory processes. Hence an inflammatory theory of the etiology of tumors is not a hypothesis.

The dyscrasic, embryonal, spontaneous, and nervous theories, as ingenious as they are, can hardly, at present, be regarded as theories, but must be called hypotheses; for so far not a single tumor can be proven to have really developed from the causes promulgated. I gave those hypotheses due consideration, but I did not attempt to criticise them for want of time and space; and, again, there is *nothing to be disproven, where nothing is proved.*

I will now enter into some details of the inflammatory view, the one I have adopted.

It is properly held by some that no line of distinction can be drawn between true tumors and chronic inflammatory products. I shall bring forward some facts now generally acknowledged, and also some investigations of my own, which will yet considerably strengthen this view.

Practically we do not know what inflammation really is;

we know only some of its causes, symptoms, and some of the terminations. But the inflammatory process itself and some of its terminations have been pretty well studied and are well known through the labors of Virchow, Cohnheim, Stricker, Ziegler, Ranvier, and Samuel abroad, and W. F. Norris, Woodward, and E. O. Shakespeare in this country. From these observations we also learn that many and sometimes all the signs of inflammation may be wanting in that process, and really the symptoms are altogether absent in many of the so-called chronic inflammations.

Many of the products of inflammation are not only difficult to distinguish from tumors, but are really recognized as true tumors.

The criterion of true tumors is regarded to be their tendency for permanency in contradistinction to inflammatory products which tend to disappear ; but it can be shown that, while true tumors occasionally do disappear, inflammatory products, very frequently, never disappear.

There are many cases of sudden and gradual disappearance of tumors on record. I shall mention only a few.

Dr. Th. Dwight[1] reports a case of an unmistakable tumor of the rectum, which had disappeared spontaneously. In the discussion of the paper the argument was brought forward, that of all tumors, only lymphoma is known to disappear, and as this tumor was single, it probably was not lymphoma, the latter tumor always occurring multiple ; the probability being more in favor of its having been medullary sarcoma (commonly mistaken for encephaloid).

Prof. Louis A. Duhring[2] has met in his practice a peculiar tumor, which he has called *inflammatory fungoid neoplasm.* This appeared suddenly as round or oval, circumscribed,

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[1] Dr. Th. Dwight, The Disappearance of Tumors, *Boston Med. and Surg. Journal*, 1880, p. 562.

[2] Duhring. See supplement to a case of Inflammatory Fungoid Neoplasm, by Louis A. Duhring, Philadelphia, 1880, pp. 12, 16, 18.

nodular or fungoid growth of a dark-red color and of the size of a pea to that of an egg. Having attained a definite size, as a rule, these growths would soften, diminish in volume, and undergo, sooner or later, spontaneous involution without pigmentation and without scar. Although resembling sarcoma, as Dr. Heitzmann of New York pointed out, Dr. Duhring considers that this disease is unquestionably an inflammatory new-formation, allied to sarcoma, but differing from it as described by authorities. The great peculiarity of these growths was their rapid rise and fall, rising in a night and disappearing within a week.

H. Fisher[1] records a case of a man from whom a large tumor of the neck had been extirpated; two days after the operation a swelling of a gland as large as a fist disappeared during a night; at the same time high fever set in and the patient died. Three days later, at the *post-mortem*, no cause of death could be found. In the axilla was found a small swollen gland. Fisher believed that a very acute fatty degeneration and re-absorption of the tumor elements took place. In a second case a tumor of a lymphatic gland as large as a fist diminished to the size of a small apple during the progress of an acute meningitis and tubercular pericarditis. In a third case during ilio-typhus in a girl sixteen years old, a lymphatic tumor of the neck, 5 cm. long and 3 cm. wide, diminished to the size of a bean. In a fourth case a goitre disappeared during the progress of scarlatina. The author adds a few cases of tumors in which, after trifling interference, noticeable diminution of size occurred.

Berns, of Amsterdam, also records similar instances of the disappearance of tumors.

Lücke[2] also observed tumors diminish and even permanently atrophy under the influence of exhausting diseases.

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[1] *Deutsche Zeitsch. f. Chirurgie*, xii, Heft 1 und 2, 1879.

[2] Lücke, *l. c.*, p. 16.

Virchow[1] says that warts, condylomata, and even fibroma have frequently been observed to heal and to disappear, undergoing a slow atrophy and resorption.

Simon[2] records the case of a recurrent fibroid which disappeared completely when treated by cold (?).

Prof. Wm. Goodell[3] says that fibroid tumors when affecting the womb at a period near the menopause, frequently undergo retrograde change. "The climacteric once reached, these growths generally grow smaller and may even disappear."

Even Rindfleisch, in his text-book, records that pediculated tumors have fallen of their own accord, and that entire cancerous nodes have been observed to cast off spontaneously.

Drs. Ripley[4] and Robinson, of New York, each recorded a case of complete disappearance of epithelioma. Dr. Robinson's case was one of epithelioma of larynx, which had perfectly healed. Dr. Ripley's case is particularly interesting, as it formed the subject for discussion in the Pathological Society of New York. The case was epithelioma of lip of several years' duration, and was not removed on account of the bad health of the patient. Subsequently the new growth spread by extension to both submaxillary glands. While the secondary deposits below the chin continued to grow, producing tissue destruction, the original growth on the lip healed and was fully replaced by perfect, healthy scar-tissue. In the discussion the following points of interest were brought forward by Dr. Satterthwaite and others: Cancers occasionally heal spontaneously, or a cure is accomplished through an acute inflammatory process in-

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[1] Virchow, Geschwülste (*l. c.*, p. 359).

[2] Simon, Discussion on Cancer, *Brit. Med. Jour.*, 1874.

[3] Goodell, Clinical Observations on the Radical Treatment of Fibroid Tumors of the Womb. Transactions Med. Society, State of Penna., 1880.

[4] Ripley, Epithelioma of Lip—Spontaneous Healing of the Original Lesion, *N. Y. Medical Record*, July 16, 1881. This is only one of the several cases which I have seen recorded since the reading of this paper.

duced by means of local applications; the healing being a process of cicatrization. The connective tissue proliferates, "squeezing all the cellular elements (of the cancer) to death," and forms dense scar-tissue.

I would like to remark here, that the healing process of an ordinary granulating ulcer is precisely the same as in cancer, wherever healing of the latter occurs. Here, like there, healing is accomplished by the additional formation of connective tissue, *i. e.*, cicatrization. Poultices, pressure, etc., act beneficially in the healing of ulcers, only because they promote the transformation of granulations into scar-tissue, and induce a fatty degeneration of superfluous cell masses; they assist the connective tissue in "squeezing to death" the exuberant granulations, from which to sarcoma there is only one step. Lücke[1] says that sarcomata in very young individuals occasionally grow as rapidly as acute abscesses, and are frequently mistaken for the latter.

I mention this in order to point out the close analogy that exists in the termination of tumors and that of inflammatory products.

We have seen that *tumors occasionally heal and disappear*. On the other hand, *it is well known that many inflammatory products*, particularly chronic ones, *never do disappear, and that the symptoms and cause of them are frequently less obvious than in the case of tumors.* The connective tissue which, in proliferating, constitutes the main bulk of elephantiasis and of the cirrhosis of organs and a good many other pathological tissues outside of tumors, never disappears.

Virchow properly considers elephantiasis Arabum and soft fibroma morphologically and etiologically identical, and in the same sense he does not admit any difference between the connective tissue of an advanced cirrhosis of

[1] Lücke, Die allg. chir. Diagnostik d. Geschwülsten, *Volkmann's Klin. Vorträge*, 97, 1875.

organs and that of a diffused fibroma. In fact, we are only in the habit of calling a proliferation of connective tissue in the mamma an intercanalicular fibroma, because the connective tissue affects an external part, while a similar affection of the liver or kidney we term an inflammatory one—a cirrhosis. Why should we make such a distinction?

I fully believe in an *acquired predisposition* to tumors. Acquired through *external influences*, *i. e.*, through any thing that may excite an inflammation or a long-continued irritation, and consequent disturbance in the tissues, *e. g.*, injuries, long standing, pressure or irritation, colds, etc. Injuries are properly regarded as exciting causes of tumors, but this may only be so in a certain class of cases, perhaps in hereditary tumors. From my inquiries I am inclined to believe that the inflammatory process creates conditions in the tissues which directly, and more than any other cause, predispose to tumor formation, and hence I would regard inflammation a predisposing rather than an exciting cause. Good and exhaustive statistics should be made in this direction.

Any inflammatory process, due either to external or internal injury or irritation, etc., may produce a new formation of tissue—a tumor. This may depend particularly upon an imperfect process of healing, as I shall show later.

We even do not need here to take into consideration gummata, tubercle, lupus, etc., the well-established products of inflammation, which so frequently occur as well-defined tumors. I think we can come to a satisfactory conclusion on the inflammatory origin of the true neoplasm even without them.

My own experience is limited, but in the cases of tumors in which I had the opportunity to get the history myself, or where I insisted upon an exhausted anamnesis in cases of others, it was possible in nearly one half of the cases to

trace out a local inflammatory process preceding the tumors at some time or other. Sometimes it dated years back. Careful inquiries nearly always revealed some cause, viz., an injury, long-standing irritation, mechanical or toxic, or an impaired or excessive use of the part, pressure, or a long-standing catarrh, or something of that nature.

Tissues which are most liable to be the seat of inflammation, are also the most *common seat* of tumors. Again, those tissues which do not participate in active inflammatory processes (ganglionic and striated muscular tissue) seldom or never give rise to tumors.

The extensive and careful statistics of Dr. d'Espine, of Geneva, show that the os uteri and the stomach are the most frequent seats of primary cancer, and they are also distinguished for their remarkable liability to catarrhs. Virchow has repeatedly pointed out in a catarrhally inflamed gastric mucous membrane the gradual transition to carcinoma.

Dr. J. H. Musser directed my attention to the fact that primary cancer of the gall-bladder is nearly always preceded by gall-stones. He demonstrated a beautiful specimen of recently developed cancer of the gall-bladder to the Pathological Society of Philadelphia, in which the clinical history revealed gall-stone for years. In looking up the literature, Dr. Musser found numerous cases of primary cancer of gall-bladder, and every one was preceded and accompanied by gall-stones. Unquestionably in all these cases the stones excited a catarrhal inflammation and this produced the cancer. A gradual transition from catarrhal inflammation of the mucous surface of the gall-bladder and duct to cancerous formation, was distinctly demonstrated in the microscopic preparations from Dr. Musser's case.

I have on several occasions contributed[1] to prove, that

[1] See Transactions of Path. Society of Philadelphia.

most of the so-called indolent ulcers are epitheliomata; nearly all those everlasting ulcers are surface cancers. But at one time they were little sores and were produced by an injury. There are a number of these indolent ulcers in Philadelphia hospitals; they are all due to inflammation, which is directly traceable. I examined many of them microscopically, and nearly every one proved to be an epithelioma.

Dr. S. W. Gross[1] is of the opinion that cancer of breast may result from ordinary eczema or psoriasis of the nipple, just as epithelioma of the tongue may follow ichthyosis or hyperplasia of the epithelium of that organ. Dr. Gross finds from his own statistics, that non-carcinomatous tumors, too, have been traced to a trauma in one example out of every eight and a half cases.

Dr. A. G. Gerster,[2] presented recently to the Pathological Society in New York three specimens, which illustrated beautifully the traumatic origin of cancer. The first case was a cancer of the sole of foot, which had killed the patient by metastasis of the growth to the brain and nearly all other inner organs. The doctor had observed the case for years, and had traced with absolute positiveness the primary tumor of the foot to a simple erosion of the skin from stepping on a nail. The second case was cancer of the outer malleolus, also directly formed at the seat of injury. The third case was a cancer of the lower extremity, developed directly in a scar, the result of a burn dating thirty years back. The tumor did, however, not develop until a year prior to the amputation, when he had struck himself accidently upon the same spot.

I have seen, myself, several similar cases of tumors positively of traumatic origin in the University Hospital clinic and elsewhere. As some of them are and others will be

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[1] Gross, Tumors of the Mammary Gland, 1880.

[2] Gerster, Specimen illustrating the Traumatic Origin of Cancer. *N. Y. Medical Record*, July 16, 1781.

recorded in the proceedings of our Pathological Society, I will refrain from mentioning them here individually.

Winkel,[1] who investigated exhaustively the etiology of myomata of the uterus, came to the conclusion that these tumors are caused either by direct excitants, viz.: coition, injury, abortion, rough removal of placenta, cellulitis; or indirectly: through repeated lifting, shock, sudden hyperæmia, etc. "These," he says, "inevitably first produce disturbance of circulation, stasis and wandering out of white blood corpuscles, etc." What do we need more; is it here not plain that the inflammatory process was the causative factor of the new growth? The author, however, unnecessarily adds: "This extravasats or transudats gives the impulse for the new-formation like an ovulum, etc."

Epithelioma of lips, one of the most common tumors, gives also a clinical proof of the inflammatory theory; here the irritation by tobacco-juice, as well as the pressure of the pipe, must be the cause, as the new growth occurs preeminently in men who are inveterate smokers.[2] I examined the teeth in three cases of epithelioma of the tongue; in every case they were bad, many being broken, and had been in that condition for years; probably the irritation and injury to the tongue were the cause of the new-formation. Similar observances have been made by others.

Epithelioma of penis has repeatedly been traced to a congenital or acquired phimosis, a condition which naturally gives rise to constant irritation and usually calling forth an inflammation.

The workmen in coal-tar and paraffine manufactories suffer very frequently from acute and chronic inflammations of the skin. Volkmann[3] has already described several

[1] Winkel, *Volkmann's Samml. Klin. Vorträge*, No. 98.

[2] I once saw an epithelioma of the lip in an Irish woman; upon inquiries I learned, however, that she had indulged in smoking a short pipe for many years.

[3] *Volkmann's Sammlung Klin. Vorträge.*

cases in which true epithelial cancer was developed from those chronic inflammations, and Tellman[1] now adds another of the same nature, ending fatally after numerous operations. This form of cancer has a parallel in the chimney-sweeper's cancer.

Most of the myelinic neuromata occur only in amputated stumps, developing at the cut ends of nerves, and hence are direct inflammatory products. (Perls.)

Frequently warts, nævi, and keloids,[2] through interference which sets up an inflammation, increase and multiply, and even are converted with malignant tumors.

Lücke and Virchow found that whenever an autopsy revealed cancer or any tumor of stomach or œsophagus, the clinical history nearly always revealed "*drunkard*." We have seen before that from long-continued catarrh to carcinoma there is only one step.

Lipomata very frequently occur in portions of the body which were subject to excessive pressure or irritation. Probably, however, we must first have the development of connective tissue,—a fibroma,—before we have a lipoma.

Extremely frequent is the occurrence of sarcoma in young persons in consequence of direct injury, or developing in any imperfectly healed scars. Hundreds of cases of chondroma and osteoma, too, have been traced by a distinct and clear history and evidence directly to blows, fractures, cuts, and other injuries.

Any one can convince himself of the above-mentioned facts by just looking carefully over the literature, and by taking careful histories of his own cases. Hundreds of tumor cases of positively traumatic origin are also recorded in the classical works on tumors of Virchow, Weber, Müller,

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[1] Tellman, *Deutsche Zeitschr. für Chir.*, vol. xii.

[2] Concerning keloids I would like to remark that, as is well known, most of them consist morphologically of cicatricial tissue. Surgeons who remove them find that they always return. They do not return, but the *scar-tissue* returns—as in loss of substance true skin is never reproduced, but only a scar.

and Broca. All the present younger working pathologists in Europe are in favor of an inflammatory origin of tumors, though none of them expresses himself definitely; still they return gradually to the view which the fathers of pathology held originally.

Inflammation is the only factor which has been traced to be the positive cause of tumors in a number of cases. This is proven by high authority and statistics. But as these authenticated cases of inflammatory origin are in moderate number, and as those with no cause, by reason of careless note-taking, are in enormous majority, the inference is drawn that inflammation has little or no significance in the pathogenesis of tumor.

I beg leave to argue as follows: In a certain number of cases it is positively known that inflammation preceded and was the cause of the new growth. In regard to the remaining cases of tumors we know nothing, no positive cause could be traced. Hence I think it logical, for the present, to consider inflammation as the cause of tumors in general.[1] All other alleged causes are only speculations; and nothing reasonable can be brought forward against the inflammatory theory. Speculations are valueless, I think, in the presence of positive facts, even if these be few in number. In science any amount of negative results are always disregarded in the presence of even a few positive facts. *Until contrary proof be given we are at present, by a mass of evidence, forced to the conclusion, that tumors represent merely one of the terminations of inflammation.*

The question now arises in what way does inflammation produce a tumor, and why and when does a tumor develop after an injury? Why is not every injury followed by a tumor if inflammation is the cause? Prof. Maas'[2] ingenious

[1] I would exclude here only the purely congenital new-formation, *e. g.*, simple angioma, and lymphangioma, rhabdo-myoma, the dermoid cysts, and a few of the nævi. These are simple congenital anomalies of the organism.

[2] Maas, *Berliner Klin. Wochenschrift*, No. 47, 1880.

answer was, that it depends upon the presence or absence of Cohnheim's supernumerary embryonic cells at the seat of the injury. If those misplaced or aberrant cells happen to be present in a part, a trauma will induce inflammation followed by a tumor; if no extra cells are present, a simple inflammation will follow, and nothing more. But this is only a hypothesis, it cannot be demonstrated. Embryonal (fœtal) cells could not continue to exist unchanged in the adult individual; nor do they need to be pre-existing in order to form a tumor. They can be and are always created by any inflammatory process.

I will try to answer the above question by facts, which microscopic examination reveals, and which will show that the study of histogenesis must go hand in hand with that of the etiology, and possibly might disclose the mysteries of the cause of tumor.

It is true that not always direct observation of active pathological processes can be made. In the case of tumors only inferences of previous cell activity can be drawn from the microscopic picture; but the pathological process can frequently be traced out under the microscope, from the various transitional stages of the elements of the new forming or formed tissue.

It is in accordance with the modern views to say that every tumor has its strict physiological prototype. Even for the cancer, only the peculiar atypical arrangement of the cells remained a criterion, while the cells themselves are supposed to be strictly identical with those found normally.

It appears to me, and the more I study the histology of tumors the more I become convinced, that any variety of cells composing a tumor are not identical with those found normally, but resemble those met with in chronic inflammatory products. In tumors, the shape and the peculiar varia-

tion in size of the cells and nuclei, the character of the intracellular network, and of the amœboid motion of certain cells, the intercellular substance, the occasional arrangement into nodes, the relation to reticulum and blood-vessels, and the peculiarity of the latter are all precisely like what is found in chronic inflammatory products and not like in normal tissues.

There is a great difference between the tissue elements of fibroma and those of normal connective tissue, for example.

I shall give briefly the details of my investigation of the structure of fibroma which, when completed, will be published and illustrated elsewhere.

Concerning the structure of normal connective tissue, the following seems to be generally established and in good preparations quite demonstrable.

The ultimate connective-tissue fibrils (the fibrillar variety) are in varying number united together to form bundles; these again occasionally unite to form larger bundles ; these bundles arrange themselves at different localities in various manner, *i. e.*, parallel as in tendons, or as a lattice work in membranes, or decussate at different angles and in all possible directions in all other localities, leaving between small spaces, these spaces being dependent for their shape and size upon the arrangement of the bundles. They communicate with one another, and thus form a system of channels throughout the whole connective-tissue system of the body. These channels contain a small amount of fluid containing *mucin*, and they are the receptaculi of the sometimes enormous quantities of serum in œdema. These same spaces or channels may also get filled with air, producing emphysema in skin and other parts of the body.[1]

[1] The subcutaneous tissue of the whole body can be filled with air, so as to produce enormous emphysematous disfiguration, by forcing air through blow-tubes at a few points or possibly even from only one point of the body below the skin. I have seen children purposely prepared in this way for beggars' purpose.

Von Recklinghausen has shown that the spaces in the connective tissue communicate with the lymphatics, and he calls the spaces juice-channels; they act as "vasa serosa" (Orth), conducting the serum from blood-vessels to the lymphatics, and "feeding" (Tyson) the tissues.

By the nitrate of silver method, of von Recklinghausen, which is now the common property of all the laboratories of the world, it can be easily demonstrated that each of the connective-tissue bundles spoken of is surrounded by a distinct membrane composed of large flat cells. These flat, so-called endothelial cells are very thin, nucleated, and are closely united at their periphery with one another, so as to form continuous membranes or sheaths, which envelop each or several fibrillar bundles, and thus at the same time form a lining for the spaces between them. Without nitrate of silver the endothelial cells cannot be seen; all that is seen are the nuclei of the cells: round or oval in shape if viewed from above, or spindle-shaped if the whole cell is seen in profile. I will not enter into further details here; this suffices to make myself now intelligible concerning some points in the histology of connective-tissue tumors, particularly fibroma.

I investigated by the nitrate of silver method three specimens of fibroma: 1st, a small, hard fibroma from the finger of a girl, æt. 20, developed from the tendon; 2d, one of the size of two fists, from the broad ligament of a woman, æt. 35; and 3d, an intra-uterine fibroma of the size of one fist, from a woman, æt. 40.

I might say at the outset that in the preparation of the first and third specimens I failed altogether to discover any perfect endothelial sheaths surrounding the bundles of fibres, which were so beautifully seen in a preparation of tendon, made for comparison simultaneous with the fibroma specimens. In specimen 2d only a few

perfect endothelial sheaths were visible. The microscopic picture of one of the silver preparations (from specimen No. 1) was this : The fibrils were on the average much thicker than in normal connective tissue; some running straight, others rather wavy and not quite parallel with one another, frequently decussating. Only few perfect fasciculi or bundles of fibres were seen, but most of them had not a trace of endothelial ensheathment. Some had a partial endothelial sheath in some places, and here the bundles appeared constricted. In several places were seen irregular protoplasmic masses apparently in connection with the fasciculi and proved to be partially detached endothelial cells. Between the bundles were seen several groups of young indifferent cells, resembling white blood corpuscles. Other cells were double the size of the latter, some spindle-shaped and with prominent nuclei. The latter were seen occasionally in a state of division, or were already divided. They resembled remarkably the germinating endothelial cells from serous surfaces, as described by E. Klein, of London, represented by him in his Atlas of Histology, plate vi.

I interpret the microscopic picture as a whole thus: The endothelial cells composing the sheaths of bundles of connective tissues have become isolated, and hence the sheaths are destroyed. The boundaries being removed, the liberated connective-tissue elements grow with great vigor. The growth is perhaps promoted yet more by the presence of the serum of the juice-channels, with which the cellular and fibrillar elements now come in direct contact, the sheaths being destroyed. The cells and fibres here, like in elephantiasis, "feed" (as Prof. Tyson would say) upon that serum in which they are soaking. The endothelium is proliferating (germinating, Klein) and probably gives rise to those groups of indifferent cells, which evidently form the main

source of the new growth. Foerster[1] has pointed out that in the development of fibroma the fibres arrange themselves more or less concentrically around and develop from these islands of cells, thus giving rise to the lobulated appearance of this new growth. It is also very probable that emigrated white blood corpuscles assist in forming those collections of cells.

What interests us at present, however, is the absence of the endothelial sheaths in the connective-tissue bundle in the fibroma, and that this feature fibroma has in common with all connective-tissue formations which owe their origin to inflammation, as will be shown directly.

I can affirm the absence of endothelial sheaths in the new-formed fibrillar connective tissue as met with in cirrhosis of organs, which invariably accompanies the proliferation of the alveolar connective tissue in such situations. It would be very desirable that other histologists would undertake research in this direction.

Cornil and Ranvier[2] describe the disappearance of the endothelial ensheathments in connective tissue which is the seat of inflammation. They describe the appearances as follows: "The fasciculi are smaller, less distinctly fibrillar; they do not appear to be enveloped by a special layer which limits them and which causes them to swell irregularly when acted upon by acetic acid." C. and R. consider that the "large flat cells" are replaced by embryonic tissue.

The inflammatory process is, to my knowledge, the only factor which can disconnect or isolate endothelial or epithelial cells united together to form a certain lining or covering. Let us take, as an instance, the lung. The flat cells which form the lining of the air-vesicles, are so closely

[1] Foerster, Atlas der mikroskopischen und pathologischen Anatomie, Leipzig, 1855.

[2] Cornil and Ranvier, A Manual of Pathological Histology, translated by Shakespeare and Simes, Philadelphia, 1880.

united or grown together in the normal adult individual, that no means at our command at present can isolate them. But in catarrhal pneumonia the inflammatory process demolishes that lining instantly; the cells which compose it "return to their embryonic state" (Stricker); they become completely isolated.

The abnormal increase in bulk of tissue in both the fibroma and the inflammatory connective-tissue products, appears to me to be due to the same cause:

1. The removal of the boundaries which keep the fibres intact, viz., the destruction of the endothelial ensheathments.

2. The proliferation of the endothelial cells of these destroyed sheaths and of the connective-tissue elements themselves, and probably with the aid of white blood corpuscles.

If the endothelial sheaths of the connective-tissue bundles and other normal boundaries are re-established in the inflamed tissue, then it will return to its normal state, or in case of loss of substance, will heal by permanent scar-tissue. The healing process was perfect.

On the other hand, the same tissue will give rise to a fibroma if this healing process was imperfect; *i.e.*, the endothelial ensheathments are not re-established, the connective-tissue elements remaining freed from any restriction proliferate on their own accord, grow above the physiological limit, and thus inflammation terminates in a tumor.

Hence, from histogenetic grounds, I would suggest that *fibromata should be classed as a product or rather as one of the terminations of inflammation.*

This is also in accord with clinical experience.

Now, is an inflammatory origin less evident in other tumors? Can there be shown any positive microscopic difference, for instance, between a mass of inflammatory granulation tissue and a sarcoma? There cannot. To my knowl-

edge distinguished histologists have repeatedly had sad experience in this.

If the discoveries of Classen and Woodward should prove correct, we would, to my mind, have another additional proof that cancer is only one of the terminations of inflammations. I will quote the following:

Woodward[1] says: "My own studies of thin sections led me to the conclusion that the migration of white blood corpuscles played a great rôle in the development of cancerous growths, and that at least in certain cases the cancer cylinders were formed by the transformation of these corpuscles, which first accumulated in the lymphatic capillaries and the passages leading to them."

Classen[2] is even still more positive, saying that he has proven "that the cells of cancer cylinders and all the elements of cancerous growths are no other than migrated white blood corpuscles escaped from the blood-vessels."

Though in my own research I did not succeed as yet to confirm the observations of Woodward and Classen, they are possibly correct, and I utilize them as coming from such high authority. Besides, they correspond so remarkably to what I believe to have established for fibroma.

My view of the histogenesis of fibroma holds good also for primary glandular carcinoma.

The glandelemma or basement membrane in glands (wherever such exists), upon which the epithelial cells rest, may be destroyed in precisely the same manner as the endothelial sheaths of the fibrillar bundles. This is demonstrable in carcinoma beginning to develop in a gland, or in the transformation of an adenoma into cancer. Here, as in fibroma, only an inflammatory process can accomplish this destruction of the normal boundary. If this boundary

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[1] Woodward, On the Structure of Cancerous Tumors. Toner Lectures, Washington, 1873.

[2] Classen, Ueber Cancroid der Cornea, etc. *Virchow's Archiv*, Heft. 1, 1870.

be not re-established after an injury, by perfect healing, there is nothing to prevent the epithelial cells from travelling into surrounding connective-tissue spaces and to thus form a cancer.

It is not the want of resistance of the surrounding tissue (as is generally held), but simply the getting loose of the normal cells from their place of attachment, which constitutes the formation of a malignant tumor.

It is the mobility of the cells, I think, that conditions the malignancy of a tumor. Any tumor, even the most benign lipoma, would be eminently malignant, if the cells composing it could get loose and travel through the widely open paths of the system of juice-channels.

It would appear that I have deviated from the scope of my subject; but I think all these points considered have a direct bearing upon the etiology of tumors. Of course, I consider this communication nothing more than an attempt at the solution of the etiology of tumors. I hope it may suggest some thoughts, and encourage others to undertake research on this subject, which, I believe, will establish the fact that *all tumors are products of the inflammatory process, and that they should be considered as one of the terminations of inflammation.*

EDITORIAL DEPARTMENT.

TREATMENT OF STRICTURE OF THE MALE URETHRA.

One can well begin a task of this nature. and upon this subject, by quoting from Pope, who wrote :—

> "What dire offence from amorous causes springs,
> What mighty contests rise from trivial things."

While it is not fair to presume that Pope had in mind each of the peculiar physical infirmities and so-called punishments often meted out to those who violate a law of God to gratify the "passions of men," yet either from practical observation, personal experience, or prophetic vision, he set to rhyme a sentiment that will appeal to the reason of the erring multitude and its medical advisors.

Had he lived till the present time and viewed with indifference and unconcern the presence of a stricture of his own urethra, while indulging in the so-called good things of the world, he would have, no doubt, like ordinary mortals, been forcibly reminded of the truth of the stanza of his contemporary John Gay :

> "So comes the reckoning when the banquet's o'er—
> The dreadful reckoning and men smile no more."

It is not impossible nor entirely inconsistent to suppose that, Pope, having had "urethral irritation" which had "from amorous causes" sprung, visited the specialists of his day and had been edified or annoyed by their individual theories and personal animadversions, rather than relieved by their art :—a vari-

ety of experience that cannot be said strictly to have died with Pope—or any one else. This fancied experience might have caused him to have written "What mighty contests rise from trivial things." However this may be, it is a fact that there is scarcely any, if any surgical condition to which the male portion of the human family is so strongly predisposed, by reason of its indiscretions, as to stricture of the urethra. Gonorrhea and other morbid processes which depend upon impure or intemperate sexual intercourse are of the most common occurrence. Any one attached to the venereal service of a dispensary or hospital can recall without any effort the coming of the aged mendicant, who with a feeble frame and tremulous voice presented his "running" for treatment. It is not of infrequent occurrence that the other extreme of life presents itself for treatment; children yet in their 'teens, children in arms, and, one might add, babes at the breast, who have become affected by the lascivious dalliances of diseased nurses. The extremes suffer the least of all; between them, in all stations of life, honor, and power, is to be found the material that fills the morning hours of the busy practitioner, as well as the venereal wards of the hospital, the class at the dispensary, and, worst of all, fills once happy homes with distrust, disease, and death.

The influence of the sexual passion is exceedingly powerful and reaches everywhere. It has caused the overthrow of empires, destroyed cities, disrupted social order, and scandalized the holy calling; all yield to its influence, and a multitude revel in that with which God endowed animal nature for the preservation of species. A prominent surgeon and specialist of venereal diseases was once heard to say, "Show me a man who has never had the clap and I will show you a curiosity." He might have added—or a very lucky man. This statement is, of course, somewhat hyperbolical, yet it emphasizes with full force the generally accepted state of things. A disease of such common occurrence, one which dates its birth so near to the inception of the human passion which predisposes to it, must cause much suffering, and be followed by troublesome and dangerous sequels. The sequel that will engage

our attention at this time is commonly and scientifically known as stricture; and, while the laity do not comprehend the full force of the word, its suggestion will often lead to a confession of judgment.

It will be our purpose at this time to recall as far as practicable, the various methods which have been employed for the treatment of stricture during the last thirty or forty years, as well as to cursorily mention their association with much earlier periods.

During this period, many of the best surgeons and most noted specialists have devoted their earnest efforts to a solution of the questions associated with it. One who attempts to follow out all of the methods, with their variations, will find himself engaged in a task not at all consistent with the time and patience of the reader, or the space to which it can be allotted reasonably in a medical journal. The classification of stricture—upon which the treatment so largely depends—has changed but little during the last half century. We meet to-day, as then, with the organic and inorganic, the traumatic and idiopathic, the congenital and acquired, the inflammatory and spasmodic forms; yet, while the classification has remained substantially the same, differences of opinion have arisen and continue to arise regarding the exact relation of one form to another, and their relations to the urethra. Their pathology, which is closely interwoven with their classification, and upon which it so markedly depends, has been a matter of dispute, especially regarding the spasmodic and congenital varieties. The treatment of to-day admits of nearly the same subdivisions as formerly, having gained, however, a more technical nomenclature, but still bearing strongly the ear-marks of the older masters. Some of the former methods are almost obsolete in practice, and are only mentioned in the text-books to be condemned. The improvements in the implements for the treatment of stricture have, if any thing, been in the advance of the results of the treatment; yet, these improvements have rendered it simpler, more satisfactory, and, in most instances, safer for the patient. The treatment is rationally divided into constitutional and local, the former usually being medicinal. While the importance of each is

well understood, the former is, at the present day, overshadowed by the latter. This may be due in part to the unwillingness of the patients to submit to delay. We think, however, that the desire of many surgeons to utilize the latest instrumental improvements has much to do with it. Still, that less emphasis is given to the general treatment than formerly, is seen by referring to *Guy's Hospital Réports*, April, 1840, wherein Mr. Bradsby Cooper said substantially : " Mechanical application contributes but little toward radical cure, unless constitutional means be combined." He still further emphasized his belief in the importance of the general treatment, when he asserted that " constitutional remedies alone may cure recent strictures." He advocated the warm bath, bleeding, opium, belladonna, etc., for irritable and bleeding strictures. If Mr. Cooper had possessed the means at the command of modern surgeons, he would have relied less, no doubt, upon the constitutional ones. The local measures had the same general classification as at present, only being necessarily less diversified. The various modes of dilatation were employed according to the peculiar views of the surgeon. It was the practical failure of this method in many cases, which naturally suggested the necessity of additional means of relief, and led to the development of the methods of treatment by cautery, divulsion, incision, etc., all of which, it will be seen, tended to hasten or supplant the absorptive and dilating influences induced by the older methods; one of which—dilatation—has borne the test of experience longer than any other, having been employed by Galen to remove "carnosities."

There are many surgeons who now rely entirely upon some form of dilatation. This is due largely to its perfect safety, as estimated by their timidity of the prompter means of relief. Sometimes, in a small and almost impermeable stricture, filiform bougies are allowed to remain a definite time, provided they cause no discomfort to the patient. This treatment, however, is not persisted in. Sir Benjamin Brodie, who is said to have spent one year in passing an instrument into the bladder, once advocated the use of continuous dilatation under the following circum-

stances: "Old grizzly cartilaginous strictures which a bougie will not dilate, strictures with false passages, strictures with an irregular-shaped urethra, strictures with rigors following each instrumental introduction." Others at his time favored this method either alone or in conjunction with caustics. In the *Lancet*, March 13, 1847, is to be found the somewhat novel method of Mr. J. Goodman, who proposed what he was pleased to call the "hydraulic dilatation" method. This consisted in throwing a stream of warm water through a catheter against the stricture, which from preference should possess a spasmodic element and be associated with acute retention. Messrs. Jordan, Shellton, Adams, and Wakley also advocated it in the treatment of strictures unassociated with these complications. This method has been recommended somewhat recently by Caze, of Strasbourg, Hadden and Golding, of New York. It has attracted attention more on account of its novelty than utility. Later, Mr. Wakley (1851), with his ingenious array of guides and sliding tubes, made use of all forms of dilatation. The same can be said of Mr. Arnott, who in 1841 recommended fluid pressure through the agency of a varnished silk tube lined with gut, which, when passed through the stricture, was distended by various fluids. These methods, while showing a great fertility of resource on the part of their projectors, did not constitute a real advance in treatment, except in so far as they taught the very useful lesson of care and gentleness in manipulation. The method by rapid dilatation has been strongly advocated, though it has never met with continuous favor, and is seldom employed at the present time, except in lieu of mild divulsion, to which form of treatment it is closely allied. It is much better to divulse mildly with a suitable instrument, than to submit the patient to the rapid introduction of separate instruments, each of which must come into extensive contact with other than the diseased tissue, thereby increasing the danger from chills and other recognized complications. The process of "vital dilatation,"—after Dupuytren,—also called the "pressure method," has few, if any, advocates at the present day. It consisted in bringing continuous pressure to bear

against the stricture by means of the point of a catheter, or other suitable instrument, fastened or held in position. This was employed in the so-called impassable obstructions. A December number of the *Medical Gazette*, 1843, contains an article earnestly advocating its use. It informs us the "pressure should be continued for an hour at a time, if necessary," and "that a half dozen sittings" will enable one to "get in." Mr. Samuel Solly, of St. Thomas' Hospital, in an April number of the *Lancet*, 1856, stated that "not one case in two hundred need be cut," that "cutting in the most skilful hands is dangerous," that the "pressure treatment," catgut bougies, and constitutional means were sufficient to cure any case. Here again is taught the lesson of caution and respectful consideration for the individuality of the human urethra. Chemical agents have been employed, until late years, in the treatment of obstinate, irritable, and bleeding strictures, at various times since the fifteenth century. Antimony, arsenic, subacetate of copper, quick-lime, and other similar agents, were advocated by Lacuna, Diaz, and others; subsequently by Parè and Wiseman. John Hunter brought it into notice in England during the latter part of the eighteenth century, and recommended the use of nitrate of silver in "obstinate obstructions."

Sir Everard Home followed, and extended its use to nearly all forms of stricture. At the beginning of the nineteenth century, Mr. Whateley advocated the superiority of potassa fusa. A little later, Ducamp, Lallemand, and others employed it in France. Phillips, Wade, Morgan, and Clarke, of England, followed; they limited the extent of its use, and improved the methods of its application. During the last few years but little has been said in its favor; its questionable action, the danger attending its general use, and the advent of better means of treatment, have rendered it objectionable in theory and almost obsolete in practice. Galvanism was first used in the treatment of stricture by Crussel, subsequently by Willebrand, Wertheimer, Jaksch, Althaus, and others. Somewhat later, Mallez and Tripier announced important and unusually successful results, which, however, were not fully confirmed

by those made at Charity Hospital in 1871 by Drs. Keyes and Beard. Later still, 1874, Dr. Newman, of this city, reported quite a number of cases successfully treated by himself. This method has attracted little more than the casual notice of those not wedded to electro-therapeutics. Much of the beneficial effect attributed to its use, no doubt, arose from the mechanical effects of the bougies and electric current, rather than any electrolytic action.

The method by divulsion, or forcible rupture of the stricture tissues, had its inception in the treatment by rapid dilatation by means of sounds, etc. It prevented the injury done the mucous membrane of the organ by the friction arising from the repeated introduction of instruments, as well as avoided many of the complications attending incision. Mr. James Arnott's silk tube dilator, before mentioned, was arranged to act on this principle. Mr. Luxmore endeavored, half a century ago, to supply the need, by the use of a four-bladed expanding instrument, which could be adapted to the continuous or rapid methods. Some years after, Leroy d'Etiolles utilized this principle; then followed M. Perrève, with his two-bladed instrument. This method grew rapidly in favor, becoming markedly illustrative of that traditional broom which is asserted to always sweep clean. Even Mr. Holt, of Westminster Hospital, whose name is closely associated with this method, became its earnest advocate in 1852, notwithstanding he asserted in the *Lancet*, in 1850, that he considered it "unjustifiable to operate upon the urethra, if a catheter could be passed, in nine hundred and ninety-nine cases in a thousand." He advocated "time and caution," and had seen no case uncured into which an instrument could be passed. In 1852 we find him with a modified Perrève in his hand, which he employed commonly in accordance with certain rules and for the following reasons:

1. "The dilator being introduced in a small compass, passes the stricture with greater facility and less pain to the patient."

2. "It can be increased from No. 1 to 12 in size, without being removed from the bladder."

3. "This is attained by bringing but one instrument in contact with the bladder."

4. "The dilating tubes can do no damage; they cannot escape from between the blades of the instrument."

5. "Dilatation can be regulated in amount to correspond to the feelings of the patient, without withdrawing the instrument."

6. "The shape of the blade causes the dilatation to be gradual, notwithstanding the size of the tube introduced."

In his wake followed Voillemier, Hillman, Jackson, Fayer, Heath, and Sir Henry Thompson. In the *Medical Times and Gazette*, May, 1863, is to be found a description of an instrument devised by Mr. Thompson for the purpose of "gradual distention," as he termed it, which was to be secured at a "single sitting;" whatever this may mean, it but resulted practically in tearing the tissues asunder and producing hemorrhage, consequently causing divulsion in the accepted sense of American surgery.

There was some excuse for the misnomer applied to the instrument—"dilator,"—since the custom of the profession sanctioned it; the term divulser, as yet, not having an accepted place in its nomenclature. The part acted by American surgeons in this drama has been a prominent one. They have not only operated with rare success, but have modified and invented instruments, adapting them to smaller strictures, easier access to the bladder, better command over the distending influence, etc., etc.

The worth of this method has been variously estimated by the modern surgeons of this country. By some it was employed without much discrimination of the nature or location of the stricture; by others to those of traumatic origin, or having irritable or resilient characterestics. At the present time it is limited, if used at all, to strictures of a dense cartilaginous formation, whether due to traumatic or gonorrheal influences, together with those having marked resilient or irritable tendencies, provided they be located in the subpubic portion of the urethra. It is just to add that Dr. Agnew accepted it with reluctance, and has since abandoned it entirely. He considers it "rude and unsurgical," and cites two facts which, in his opinion, militate against its

use. (1) "Nothing can be accomplished which cannot be obtained by gradual dilatation. It is true the latter is a slow process, but I can conceive of no reason connected with a mere consideration of time which justifies a surgeon in jeopardizing the life of the patient." (2) "The tendency to re-formatiom of the stricture is not lessened by divulsion; after laceration of the tissues, an ulcer is left which can be repaired only by granulation and cicatrization; the new tissues will not become like the normal urethra, but will contract and certainly demand the repeated use of bougies for an indefinite time." Drs. Gouley, Van Buren, and Keyes, and other recognized authorities, speak kindly of the method when limited to the situation and variety of strictures before mentioned.

Incision, or cutting of strictures, is not a modern operation. According to some authorities it was practised at the beginning of the Christian era. There are definite accounts of its employment in France by Allies in 1775, by Physick, of Philadelphia, in 1795, by John and Charles Bell in 1807, and Stafford in 1827. Many of the older surgeons employed caustics in conjunction with incision. While the English and American surgeons showed due dilligence in the advancement of this method, to the French surgeons belong much of the credit of having developed it, aided by their almost innumerable and variously constructed urethrotomes. Among those who were closely associated with the method in France are to be found the names of Ducamp, Amussat, Civiale, Sédillot, Ricord, Mercier, Reybard, and Maisonneuve. To describe the peculiar method of each is impracticable and unnecessary, if not almost impossible. The subdivisions of this method are substantially the same to-day as when accepted by the older surgeons, viz., internal and external division; internal division being limited to those obstructions in front of the triangular ligament, by some, and to those in the spongy portion, by others, each of which may be incised from behind forward, or the reverse.

Something of the early history of this method of incision has already been given.

The method of external division is, as one would suppose, of older date than the internal. The latter begot the necessity of making incisions in the dark with rudimentary implements, while the former served to utilize the sense of sight and touch, as well as the ordinary cutting instruments of its time. To Wiseman, in 1652, is said to belong the credit of having first performed the operation with a view of curing stricture. A few years later Solingen repeated it. It was done by Tolet and Colet in 1690, then by Petit and Le Dran in 1740. The cases operated on by these gentlemen admitted of the introduction of an instrument into the bladder. In 1783 John Hunter, without a guide, did what is now sometimes called perineal section, but more correctly known as external perineal urethrotomy. He was, however, antedated by Molins, an English surgeon, who did a similar operation in 1662.

Perineal section was rarely done till employed and championed by Granger in 1815. In 1817 Alexander H. Stevens added his name to its list of supporters. Then followed Dr. Jamieson, of Baltimore, in 1820–23. Dr. David L. Rogers, of this city, in 1823 reported twelve cases successfully treated.

Dr. J. C. Warren, of Boston, and other New York surgeons employed and approved the method. In 1840 Dr. Syme, of Edinburgh, used it, and subsequently, 1844, forcibly proclaimed himself in favor of cutting old, tough, and resilient strictures; "those where relief can not be obtained by the passage of instruments." Many of those who preceded him, employed it only as a means of treatment in retention of urine. The claim has been made by some that to Mr. Arnott, of Middlesex Hospital, in 1822, belongs the credit of first having used it for impassable and uncomplicated stricture. Be this as it may, no one will gainsay the fact that to Dr. Syme belongs the honor of developing its worth and contending for its permanency. Associated with this operation are to be found the names of Gouley, Van Buren, Wood, Weir, and others of this city, and to these gentlemen belongs much of the honor of having offered the surgeons of the present time the means of triumphing over urethral obstructions. The

permanency of external perineal urethrotomy is well established, and in cautious hands it will serve to perpetuate the names of those who have perfected it. The subcutaneous method, advocated by Dick and others, has attracted but little attention, yet it has been and now is employed occasionally by surgeons of note; its application being properly limited to the penile portion of the organ. The methods most in use for the treatment of strictures anterior to the subpubic arch are gradual dilatation and internal incision; the former having the greater number of advocates. There is much to be said in favor of both methods; much argument and not a little animosity have been exhibited during the last few years by those who are strongly wedded to their special forms of treatment. To Dr. Otis, of this city, undoubtedly belongs the credit of having instituted something like a new era in the location, number, and size of strictures, as well as the capacity of the urethra, and the relation which it bears to the circumference of the organ. Mr. Berkeley Hill, of the University College, England, while speaking of the views of Dr. Otis in a communication to the *Lancet*, Apr., 1876, said that "Dr. Otis during his visit here enunciated views which vary considerably from, and, indeed, are opposite to the doctrines usually taught in this country." Many other allusions complimentary to Dr. Otis' independence of thought, action, and ingenuity of resource can be found scattered through the medical journals of England, and in not a few in this country.

The questions raised by Dr. Otis may be briefly summarized as follows:

1. Regarding the normal calibre of the urethra.

2. The definite proportional relation of the urethral circumference to the circumference of the flaccid penis containing it.

3. The existence of strictures larger than the previously accepted size of the calibre of the canal.

4. That gleet and troublesome reflex irritations depend upon these strictures.

5. The greater frequency of stricture in the anterior portion of the urethra than elsewhere.

6. The possibility of a radical cure by this method of treatment.

7. The advantages of this method over the methods by dilatation as regards safety, comfort, time, and permanency of results.

That the normal calibre of the urethra had not been correctly estimated, prior to the investigations of Dr. Otis, has been conclusively proven by that surgeon.

One has but to refer to the dimensions laid down by Sir Henry Thompson, which had heretofore been accepted as practically correct, to realize the misapprehensions under which he has labored. The importance of a better knowledge of the size of the urethra can but have a direct bearing upon the proper treatment of stricture. Constrictions which before this were supposed to have been distended to the normal calibre of the canal, are now found to require the use of a much larger-sized instrument, to meet the aim previously sought and supposed to have been attained.

The older masters were satisfied if a No. 8 could pass, and advised it to be "employed continuously." An exponent of gradual dilatation, at the present time, might as well stop at No. 8 as No. 16, so far as the ultimate result is concerned. If it be necessary to distend the urethra quite or entirely to its normal calibre for the practical cure of a stricture by dilatation, then the importance of determining its calibre by actual measurement or practical deductions is clearly obvious. It has been the custom of conscientious and well-informed surgeons of past time, as it is at the present, to say to a patient with a strictured urethra, "I can not assure you I will cure the stricture ; it will probably return if my directions be not followed"; which means to pass a sound as often as in his judgment the case may require. May not the necessity for these candid and humiliating statements depend largely upon the fact, that till Dr. Otis determined the actual calibre of the urethra, the treatment had not been carried to the extent of dilating the canal to nearly its normal dimensions? The surgeon might as well cease at No. 6 or 8—like our forefathers—so far as a cure is concerned, as at No. 16 or 18, as many do now, since the

latter measurements are but little nearer the normal size, of the urethra as now determined, than the former were to its supposed dimensions. It is certain that if dilatation is to be practised with a view of ultimate recovery—how else can the possibility be determined—the strictured tissues must be subjected to a far greater degree of dilatation than formerly. While we gladly admit the truth of the assertions of Dr. Otis regarding the greater size of the urethra, we are as yet unwilling to endorse his belief in the uniformity of measurement from the bulb to the meatus. We have seen meatus corresponding in size to the passage beyond, but it has been the exception rather than the rule. The capacity of the canal, in our opinion, is, as a rule, greater beyond, than at the meatus.

It has been our misfortune,—or rather the patients',—in two or three instances, to have incised the meatus too freely, which caused an imperfect delivery of the urine, soiled the clothes, and tarnished the foot-wear. In each of these cases, however, the most expansible portion of the canal, after the operation, was still beyond the meatus. It is our opinion that the meatus externus is narrower and less expansive than any portion of the canal beyond, and that this plan of construction serves an important function in regulating the expulsive power of the bladder and urethra, as well as the integrity of the stream. That a more or less definite relation should exist between the circumference of the flaccid penis and the canal it contains seems quite natural. Its recognition is an important aid in regulating the size of dilating instruments, thereby obviating the danger of over-distention. It likewise reduces an heretofore empiric procedure to one resting upon an almost scientific basis. That strictures do exist larger than the formerly accepted size of the passage, is evident when one recalls the fact that the circumference of a moderately distended urethra was estimated at from twenty-five (25) to thirty-five (35) millimetres, depending upon the portion measured, which does not correspond in size with many constrictions detected by the urethrotome. It is fair to presume, in that strictures vary in calibre, that slight constrictions of the now determined circumference will not reduce it to the dimensions previously ac-

cepted as normal. The assertion that gleet and troublesome reflex irritations often depend on these strictures is unquestionably true so far as gleet is concerned; nor is it impossible to suppose that various neuralgias and spasms may be caused, since constrictions of the passage are well known to produce them. The assertion that an organic stricture in the anterior portion of the urethra will cause a more or less persistent spasmodic contraction of the deeper part, has given rise to considerable discussion, much of which has been intemperate in its diction.

This idea was advanced by Civiale in 1850. Mr. Hancock, in the *Lancet*, February, 1852, expressed the belief that spasmodic contractions might occur at not only the membranous part but "within one inch of the external meatus"; that "these spasmodic affections frequently accompany organic strictures," etc. This question demands a careful and unprejudiced examination. It is certain that there are many cases having obstructions in the membranous part, that are associated with organic stricture in the anterior portion. It is likewise certain that these obstructions are often quite fickle in their characteristics; sometimes very rebellious, again failing to offer much, if any obstacle to the passage of an instrument. Whether these phenomena depend entirely upon a pure spasmodic contraction, incited by anterior irritation, or the contraction be caused by direct instrumental contact, or other causes, we are unwilling at present to declare. Neither are we yet prepared to accept the dictum that strictures of an acquired nature occur more often in the anterior portion of the urethra than elsewhere. We are prepared, however, to examine with care all cases which may fall beneath our tender mercies, and hope to be able to substantiate the assertion made by Dr. Otis regarding them.

If it be possible to cause a radical cure, we believe the cutting process holds out the best opportunity yet known. When properly done it divides all the contracted tissues, and the space becomes filled in by the projection of the submucous structures, and "patched" by a new growth, which can hardly possess the

same characteristics as the original trouble. This, together with the absorption of the indurated tissue, is thought to reduce the canal to its virgin state, plus a patch of new tissue.

However this may be, those who advocate the method show conclusive proof that strictures which were cut six or eight years previously, have not as yet manifested their presence by symptoms, or on exploration.

We feel that a certain proportion of them, at least, should become radically cured, since it would be but a just reward to the patient for the additional dangers incurred. We do not believe this method to be as safe as that by dilatation. The fact that the tissues are divided, that hemorrhage occurs, that inflammation must follow in the process of repair, and that these expose the patient to dangers from the recognized sequels of operations here and elsewhere on the body, seem to us of necessity to settle the question in the negative. We can recall, in our own experience, the instance of a young man with a tough stricture of the spongy urethra, which was cut. The hemorrhage was more or less constant, and often profuse, for two weeks, requiring the use of the best recognized methods to stop it. The urethra became inflamed, and an abscess was near forming; but the patient recovered with a cordee which lasted some months. No one claims to be able to say positively when or how much bleeding will occur, or when it will cease. It is impossible to accurately predicate these facts.

We have learned of cases that have been almost exsanguinated, of cases that have died from pyæmia, and other sequels. That these are exceedingly rare there can be no doubt, yet that they are liable to occur, and do occur—two undisputed facts—will always clothe the method by incision with greater danger to the patient than from gradual dilatation.

So far as time, comfort, and permanency are concerned, the method by incision outvies all others in expediency. The truth rests simply here, no one method can be used to the exclusion of all others, and whoever advocates this or that method as his exclusive one will, no matter how good the method or great his reputa-

tion, cover both with obloquy. We believe this method should be advanced with great caution, else the young, inexperienced surgeon, becoming fascinated by its brilliancy, with visions of a great reputation and a large bank account before him, may, in his sincere efforts to reduce to the standard size the male urethra, in his vanity, become a victim to that "vaulting ambition which o'erleaps itself and falls on the other side."

J. D. BRYANT.

NEW BOOKS AND INSTRUMENTS.

A New Foot Dynamometer.* By W. R. Birdsall, M.D.

If an apology is necessary in presenting what at first sight may seem too unimportant a subject for the consideration of such a society as this, the answer may be given that any means which renders more exact and perfect our method of physical examination is not unworthy of consideration.

Having felt the want of a more accurate method for determining paretic conditions of the lower extremities than the usual one by watching the gait and testing the resistance of certain groups of muscles in the hand, I attempted to construct an instrument corresponding in character to the dynamometer used in testing the power of the grasp in the upper extremity. The instrument before you is the result.

It consists of a *base-board* 18 x 6 inches, in which are mortised *two upright supports* for an *iron rod* which forms an axis on which the *foot-board* turns. *Three grooves* are cut in the base-board at one end, and *corresponding grooves* in the under surface of the foot-board, into which to slide the ordinary *elliptical spring dynamometer* used for testing the grasp. An adjustable *gauge* slides on the upper surface of the foot-board for the purpose of giving a definite position to the foot. In order to fasten the foot firmly to the board and furnish a point for traction, a broad toe-strap is used when the anterior tibial group of muscles is to be tested, and a narrow heel-strap for testing the posterior group. It is prevented from slipping by being passed through slits in the foot-board. The latter is also covered with rubber to prevent the foot from slipping.

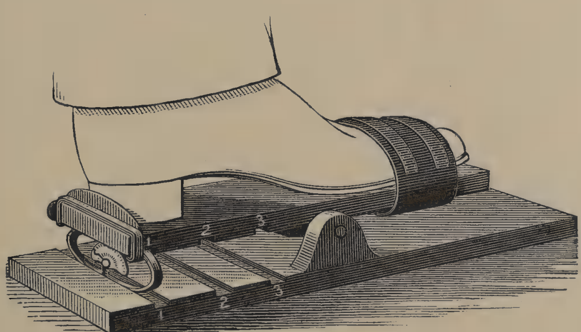
The instrument is operated in the following manner: For testing the anterior tibial group of muscles: the person being seated,

* Read before the American Neurological Association, June 17, 1881.

the foot is placed on the foot-board, in a position at right angles to the leg, which should be perpendicular. The heel rests over the spring, against the gauge which is placed in position No. 1, No. 2, or No. 3, according to the length of the foot. The toe-strap is then passed around the foot-board and the foot, and strapped as tightly as possible. The spring is placed in one of the grooves. The person is then told to flex the foot as strongly as possible. This action of rotation at the ankle joint, the foot being secured to the board, produces traction on the forward end of the foot-board and a downward pressure at its rear end, these two parallel but unequal forces tending to rotate the board on its axis, compressing at the same time the spring which offers resistance, whose indicator shows the amount of work accomplished. As the spring is not stationary, it is removed each time for the convenience of reading.

If, when the gauge is in position No. 1 and the spring in groove No. 1, a certain result be obtained, the removal of the spring nearer to the foot-board axis would give the foot a greater leverage, and, consequently, a greater number of degrees would be exhibited on the indicator; while with the spring in groove No. 1 and the gauge at No. 2 or No. 3, the reverse effect would be produced. This enables one to adjust the instrument for different degrees of foot power.

In testing the posterior group the instrument is reversed: the toe being placed against the gauge, and the heel-strap fastened over the instep, on an effort being made to raise the heel (contraction of the posterior tibial group), traction is made by the heel, and pressure by the ball of the foot, acting upon the instrument as in the previous case.



A NEW FOOT DYNAMOMETER.

The foot-board may also be strapped on to the posterior surface of the leg, and an effort made to extend the leg upon the thigh will also be indicated, though with greater chances of error than in the other cases.

As in the dynamometer for the hand, so in this one, it is not the absolute power which we particularly desire to determine, but the relation between the right and the left sides, so that the position of the foot or of the spring is not of much importance, provided the conditions are the same for each member tested. It is to secure this that the gauge was added and definite positions given to the spring.

For the purpose of uniformity in registering cases, these two conditions are indicated by a fraction in which the position of the gauge represents the numerator—being above—and the position of the spring the denominator. Thus if the gauge be back of the first groove and the spring in the first groove, the formula would read $\frac{1}{1}$; if the spring be changed to the second groove, the gauge remaining as before, $\frac{1}{2}$ would be the formula, etc.

Precautions.—1st. Be sure that the feet are in the same position on the board; this is easily accomplished by means of the gauge against which the heel or the toes rest.

2d. See that the strap is tightly adjusted so that there is no slackness. This is the most important point of all, and constitutes the greatest source of error in the use of the instrument. The excursion made by the foot in contraction of the muscles of the anterior tibial group is not great, and if the foot must be raised even a short distance before it begins to exert traction, a good deal of power is lost. I have found, however, that with proper care this can be avoided.

3d. As flexion or extension of other muscles makes considerable difference in the power of contraction, the position of the extremity and that of the instrument should be as nearly alike as possible in both feet. The proper position is to have the foot at right angles to the leg, the latter being perpendicular.

4th. Care must be taken that the weight of the body is not thrown upon the instrument. This may be avoided by a sitting posture, not allowing the subject to bend his body forward.

5th. The tests should be repeated at least five times for each foot, and the average taken; this applies as well to the hand dynamometer, for the subject in his first effort may not quite understand what movement should be made, and as it is a matter of

voluntary effort the results at all times will vary on account of unequal exertions.

6th. See that the spring is always introduced in the same position and is firmly located in the groove.

My object has been to construct a cheap and simple instrument that would not get out of order and which could be easily and quickly adjusted.

This instrument is so simple in construction that any carpenter and blacksmith can make one. As those who would use such an instrument have already a hand dynamometer, an important item in the expense is done away with.

If it is desired, the instrument can be converted into a dynamograph by fastening with a clamp a Pond's sphygmograph upon the outside of one of the uprights, and allowing a bent wire fastened to the edge of the foot-board to impinge on the rubber cap of the sphygmograph.

It is hardly necessary to dwell upon the uses of the instrument. All that is claimed for it is that it furnishes a more exact method for studying the distribution of paresis in the anterior and posterior tibial groups of muscles than those hitherto in use.

I may state that in the majority of healthy persons, I have observed very little difference in strength between the right and the left sides.[1]

Rheumatism: its Nature, its Pathology, and its Successful Treatment. By T. J. MACLAGAN, M.D. London: Pickering & Co., 1881, pp. 333.

The chief aim of the author of this work is to explain all the phenomena of articular rheumatism by means of the germ theory of disease. His procedure is logical. Before advancing his own views he attacks the lactic acid theory, the only one that has any claim to general acceptance. Confidence in this theory had already been shaken by the failure of the alkaline treatment, and still more by the brilliant results of one which, in spite of Dr. Maclagan's claim to the contrary, must yet be regarded as, in a certain sense, empirical. I refer, it is scarcely necessary to say, to the treatment by the salicyl compounds.

While denying that lactic acid is the cause of rheumatism, Dr. Maclagan admits its presence in abnormal amount in that affection, but considers it and the joint inflammation to be common effects of a rheumatic poison, which enters the system from with-

[1] Tables were exhibited illustrating the manner of recording cases and the results in certain diseases.

out. He also admits that this excess of acid may be a secondary cause of the articular and muscular symptoms, its accumulation leading to functional disturbance of the locomotor apparatus of which it is an excretory product. Functional disturbance, he says, is manifested in white fibrous tissue by pain. It is thus that he explains Dr. Foster's remarkable case of a diabetic, in whom the administration of lactic acid was followed by six well-marked attacks of an affection which it was impossible to distinguish from acute articular rheumatism. "Excess of acid may cause joint pains, but what causes the excess of acid?" In Dr. Foster's case the "lactic acid was given to the patient, and its presence in excess was readily accounted for. In acute rheumatism the excess of lactic acid is the phenomenon which, of all others, it is at once most essential and most difficult to explain." Dr. Maclagan's explanation is that the acid is the product of an unusual activity in the retrograde metamorphosis of muscle, which, considering that muscle is not generally regarded as being prominently affected in articular rheumatism, is not entirely satisfactory. Rather too much space is, in our opinion, devoted to the refutation of Richardson's experiments of injecting lactic acid into the systems of some of the lower animals. Senator treats these same experiments in a much more cursory manner. After stating that they had been disproved by Möller and Reyher, he remarks that it would have been surprising if they had succeeded, as they were performed upon animals,—cats and dogs,—almost wholly insusceptible to rheumatism, and peculiarly unsuitable for such experiments in that they are devoid of sweat glands.

A chapter is devoted to the miasmatic theory of rheumatism, the one adopted by the author. Other observers have noticed a similarity between the febrile course of rheumatism and the malarial fevers. Senator considers rheumatism allied to malarial fever and influenza, but points out, at the same time, that the maximum prevalence of the disease between October and May, militates against the miasmatic theory.

The principal points of resemblance between rheumatism and malarial fever, referred to by Dr. Maclagan, are rather negative than positive. They are as follows:

1. Both are "specially apt to occur in low-lying, damp localities, in certain climates, and at certain seasons of the year.

2. "Some people are more liable to be attacked than others.

3. "They have no definite period of duration.

4. "They are not communicable from the sick to the healthy."

He traces still further analogies between their symptoms, to which we have not space to refer.

Dr. Maclagan divides the miasmata into two classes, eruptive and non-eruptive, classing rheumatism as an eruptive fever. The distinction, says he, between an eruptive and a non-eruptive fever is, "that the latter consists simply of fever, the former of fever plus a local lesion. The local lesion of rheumatic fever is the joint affection." It seems to us that on similar grounds the splenic tumor of intermittent fever might also be called an eruption, for it is certainly a local lesion.

Dr. Maclagan's theory may be briefly stated as follows :

In all the contagia and miasmata, the poison, entering from without, finds, in some part of the body, a nidus in which the germs are reproduced with great rapidity. This nidus, also called by him the second factor of the poison, he regards as essential to the reproduction of the poison germs. It varies in amount and situation in different individuals and may, it is to be inferred, be absent in those insusceptible to the disease. When the heart is attacked, it is because the second factor exists in the fibrous tissue of the valves. All this is in strict analogy with what is known regarding the habits of parasitic organisms whose existence is not at all hypothetical, and is a logical result of the germ theory.

Dr. Maclagan quotes from the work of the late Prof. J. K. Mitchell, on the "Cryptogamous Origin of the Malarious and Epidemic Fevers," but, strange to say, appears to be entirely unacquainted with the same author's views regarding the spinal origin of acute articular rheumatism. Our space permits no more than a mere reference to the interesting papers of Prof. Mitchell (contained in the volume of the *American Journal of the Medical Sciences* for 1831, p. 35, and the volume for the same journal for 1833, p. 360), and the remark that his views have been of late years revived by the writings of Charcot and Weir Mitchell.[1]

The chapters on the different forms of rheumatic cardiac inflammation are highly interesting and, in some respects, entirely original. He denies the existence of inflammation of the valvular portion of the endocardium, except as secondary to inflammation of the fibrous structure of the valves, and when endocarditis coëxists with myocarditis, he considers that the former is secondary to the latter. In this he differs from Roberts, Bristowe, Peacock, and others, and solely on the ground that the endocardium is a non-vas-

[1] See article on "Spinal Arthropathies." *Am. Jour. Med. Sci.*, April, 1875.

cular structure. We agree with him in this view, for as the nutrition of a non-vascular structure must be secondary, so must its perversion of nutrition, known as inflammation, be also secondary. Dr. Maclagan is of the opinion that the diagnosis of myocarditis is not the " impossible thing it is usually supposed to be," and considers that such diagnosis may be a matter of " supreme importance " to the patient, but, except as indicating the necessity of absolute quiescence, this hardly appears in the sequel, for there is no difference in his treatment of cases whether they have heart disease or not. Salicin, administered so as to saturate the system as soon as possible, is the treatment of all alike. A muffling of the heart's sounds he considers diagnostic of myocarditis, distinguished from the muffling of hypertrophy by the absence of signs of increased force of action.

In regard to the method of administering salicin, Dr. M. finds that one ounce is requisite to remove the acute symptoms, and this amount should be administered within the first sixteen or twenty-four hours, after which it should be given in gradually diminishing doses for a week or ten days. His emphatic recommendation that the patient should keep his bed for one week is a most significant commentary upon the success that attends this treatment. The beneficial effect of salicin is most marked in early attacks. In chronic thickening of the fibrous textures, due to repeated attacks, exacerbations of pain may be excited by causes other than the rheumatic poison, and over these the salicyl compounds exert no effect. In such cases the alkalies are of service.

While characterizing the administration of salicylic acid in rheumatism by the German physicians as a " pure piece of empiricism," Dr. Maclagan claims that his use of salicin in the treatment of the same disease " more than a year before salicylic acid was brought into notice by Stricker and Riess " . . . . . "was not a piece of empiricism but a logical inference." He observed that a "low-lying, damp locality, with a cold rather than warm climate, are the conditions under which rheumatism is most likely to arise." He further observed that the plants indigenous to this kind of soil and climate belong to the order Salicaceæ. "Among the Salicaceæ, therefore, I determined to search for a remedy for rheumatism." The brilliant success attendant upon this search is enough to encourage others to pursue similar investigations.

Without pausing to discuss the precise meaning of the term *empirical*, as applied to the use of a drug, we would make the criticism that Stricker and Riess were probably familiar with Dr.

Maclagan's success with salicin in the treatment of rheumatism, as he had employed it more than a year before they began the administration of salicylic acid in the same affection ; and as they were certainly aware that the two drugs belonged to the same organic series, it is more than probable that their use of salicylic acid in the treatment of rheumatism was also "not a piece of empiricism, but a logical inference" from the facts observed by Dr. Maclagan.

Dr. M. combats the view of Senator that salicin owes its therapeutic virtues to its conversion into salicylic acid, both on chemical grounds and because "salicin possesses therapeutic virtues not possessed by salicylic acid ; and that salicylic acid gives rise to symptoms which do not follow the administration of salicin." Further, when a patient is suffering from the toxic effects of salicylic acid, salicin may be administered freely with benefit. Under its use the rheumatic and salicylic symptoms both disappear. These important statements are apparently confirmed by interesting reports of cases.

Dr. M. acknowledges that the hopes entertained in regard to the salicyl compounds as prophylactic against the endocardiac complication, have not been realized, and attributes this failure partly to the use of insufficient doses, but chiefly to the insidious nature of the heart affection. The endocardial blow, its first indication, is consecutive to thickening of the fibrous structure, friction of the segments, and roughening, as a consequence of such friction. Therefore, when an endocarditis is detected, it has probably existed for at least thirty-six hours. In cases of acute pericarditis, he recommends venesection, leeches, and blisters, but very properly limits the employment of the first of these measures to the "urgent symptoms of the first stage of a very acute attack."

Dr. Maclagan recognizes three conditions under which head symptoms may occur in rheumatism.

1. As a symptom of inflammation of the membranes of the brain.

2. As a symptom of inflammation of the substance and membranes of the heart.

3. In connection with very high temperature of the body.

The first is so rare that he entertains a "grave doubt" whether it be not an accidental complication. Head symptoms in the course of heart disease he regards as due to unusual nervous susceptibility ; while the third variety, cerebral hyperpyrexia, he

considers due to the irritant action of lactic acid upon the thermal peripheræ. This irritation, transmitted to the thermic centre, may result in paralysis of the heat-inhibiting function, and consequent hyperpyrexia. This conclusion he reaches after a most interesting discussion of the relations between cerebral hyperpyrexia and sunstroke.

Dr. Maclagan is evidently unacquainted with the able paper on cerebral rheumatism, by Prof. Da Costa, contained in the *Am. Jour. of the Med. Sci.* for April, 1875. If he had been, he might have referred to still another condition under which head symptoms occur. Da Costa regards albuminuria as the "most common cause of a group of the disorder mainly characterized by stupor and coma."

The arrangement of the last chapters of the book is faulty. The chapter entitled "Cerebral Rheumatism" is devoted to a discussion of the first two conditions under which head symptoms occur; then follows a chapter upon the relation of rheumatism and chorea, one, by the way, of great interest; and, finally, the chapter entitled "Rheumatic Hyperpyrexia." The order of the last two should be reversed.

The work is an infallible witness of the author's extensive and accurate knowledge of physiology and pathology, as well as his practical acquaintance with the subject of which he treats. It is a rare combination of the theoretical and the practical, and, as a whole, receives our most emphatic approval. It should be carefully perused by all who wish the latest information concerning the salicin treatment from its dicoverer, as well as by those who desire to be acquainted with the latest views in regard to the pathology of an affection which is, indirectly, one of the greatest scourges of the human race. [F. P. H.]

A Practical Treatise on Impotence, Sterility, and Allied Disorders of the Male Sexual Organs. By SAMUEL W. GROSS, M. D., etc. Philadelphia: Henry C. Lea's Son & Co., 8vo, pp. 174.

The book we are called on to review is written by one so well known to us and to the profession, on account of the valuable little work on "Tumors of the Mammary Gland," which was issued a short time since, as well as other meritorious productions, that the usual task attending a review becomes a pleasure by reason of the anticipated knowledge forthcoming. The work in question does not comprehend all of the disorders to which the male genital organs are liable, but is limited to "impotence, sterility, and allied disor-

ders of the male sexual organs." It is composed of 170 pages of well-printed matter, and contains 16 illustrations descriptive of the instruments variously employed, together with those illustrative of the various pathological conditions incident to the diseases of which it treats. Chapter first is devoted to impotence, which is defined as "an inability to copulate or perform the sexual act, due either to a deficiency or absence of the power of erection," plus "all other conditions which render the intromission of the penis impracticable." About 70 pages are allotted to this subject, wherein it is classified into the atonic, psychical, symptomatic, and organic varieties. The atonic variety is considered to be due to changes in the prostatic portion of the urethra, or to a modified reflex excitability of the genito-spinal centre, which is located, according to Eckhard, from the first to the third sacral spinal nerves. The author's arguments and conclusions are based upon the records of 149 cases which have come beneath his own observation. Space will not allow of a separate consideration of each form of impotence in detail, but it is proper to say that the best possible use is made of the cases to show that stricture of the urethra, and other morbid urethral processes bear a very close causative relation to it. About 50 pages are devoted to sterility in the male, which is defined to mean infertility or absence of seminal fluid. The relative frequency of sterility in the sexes is based upon 192 cases in which the husband and wife were both examined. His conclusions show the husband to be in the fault in one in every six cases. Napoleon the First might have been spared many of his historical protestations could he have been submitted to the test of latter-day science. Chapters third and fourth, the final ones, are devoted to spermatorrhœa and prostatorrhœa, and, like their associates, are clearly entitled to careful thought.

The general style is good. Dr. Gross is a gentleman who certainly has something of use and interest to submit when he writes a book, which fact alone is very reassuring. He chooses his subjects with good sense, treats them succinctly, and draws conclusions which his facts sustain. He is ofttimes positive, even to dogmatism, which, we believe results from a full faith on his part in the justice and truth of his conclusions. We notice a strong tendency to create new words—to extend the nomenclature of the subject—as well as to make use of those not usually met with in text-books. While there is no law against verbal proliferation, yet the simpler the text the more acceptable will be the work to the profession at

large. We willingly welcome this little volume as an useful addition, and commend it to the consideration of all. [J. D. B.]

The Diseases of Children. By WM. HENRY DAY, M.D. Second edition. Rewritten and much enlarged. Philadelphia: Presley Blakiston, 1881, pp. 752.

Dr. Day's treatise cannot be said, even in the second edition, to sum up much recent knowledge in pædiatrics. Hence, from this point of view, it is no advance upon the several excellent treatises already extant, among which Meigs and Pepper's remains our favorite in the English language. As a systematic treatise it falls decidedly behind the lectures of Henoch on children's diseases, which have just been published at Berlin. For, in about the same number of pages, it contains conspicuously less information on the subjects which it treats; it touches upon fewer subjects,—several, especially from among the diseases of the new-born, are omitted altogether; those discussed are handled in a way at once less comprehensive and less precise; more skill is shown in avoiding knotty problems than in their elucidation; finally, the personal clinical experience on which the volume is professedly based, seems to us remarkably meagre. The average clinical outline of ordinary diseases is drawn in a manner sufficiently clear and concise to be readily apprehended by the beginning student. But such outlines have often been drawn before; and Dr. Day, instead of adding to the labors of reputable predecessors, has often rather subtracted from the results obtained by them. This remark especially applies to the paragraphs on pathological anatomy, and to the grouping of clinical variations in the type of diseases,—even to those which are characteristic of childhood, and hence the special subject of consideration in a treatise on pædiatrics.

Thus we are told that in typhoid fever the "temperature runs up to 103° or even to 105°" (p. 87). But there is no hint, much less description, of characteristic temperature curves, or of their peculiar modifications in childhood. We should not, indeed, suppose, from Dr. Day's description, that this fever as seen in children differed in any particular from the ordinary adult types. The most ferocious lesions are described in the intestine. "Near the ilio-cæcal valve is shown a tendency to destruction of the mucous membrane, and ulceration or even sloughing or perforation of the peritoneal coat. The glands of Peyer's patches take on the appearance of vesicles or pustules, and subsequently they burst and produce an ulcer with oval or irregular outline," etc. Now it is a well-known fact that the enteric lesions in the typhoid fever of children are

very slight. To quote Henoch again: Out of 10 fatal cases among 137 observed, Peyer's patches were only ulcerated in 3, and in these the ulceration was slight.

Similarly, the nervous symptoms are usually remarkable for their mildness; but for this the reader is not prepared by Dr. Day. The relations of scarlatina to diphtheria are not mentioned; the tonsillar exudation in "scarlatina anginosa" is described as "yellowish lymph," and not further considered. Albuminuria, or rather the acute desquamative nephritis of which it is a symptom, is looked upon as an accident resulting from exposure to cold. But, in reality, quite apart from such exposure, the direct action of the fever poison on the kidneys is of itself sufficient to excite nephritis. The poison does not "escape" through the skin, when it has paralyzed the cutaneous circulation and thus caused the eruption. It is certainly not "escaping" at the period of desquamation; hence is not liable to be pent up in the body by a chill.

There are important varieties of scarlatinous albuminuria, or nephritis, but these are not distinguished by Dr. Day. Two useful remarks are made, however, in this connection: that tuberculosis often originates in scarlatina; and that headache occurring in children, perhaps at a long time after convalescence from this disease, should awaken suspicion of uræmia and latent nephritis.

In regard to pathology, our author rests with the opinion of Harley, "who describes scarlatina as essentially a disease of the lymphatic system." He also quotes Klein's researches, but very imperfectly: "In the kidneys there is a proliferation of epithelium cells, *and* changes in the walls of the blood-vessels. Later on there is a development of round cells, which constitute a true interstitial nephritis, due to an embolic process." Now Klein asserts that hyaline changes in the blood-vessels precede all alteration of epithelium, being observed in cases which have succumbed after only a two days' illness. We cannot understand how the lymphoid infiltration of diffuse nephritis can in any way be attributed to embolism!

Dr. Day describes acute croupous pneumonia as an ordinary disease of childhood, whereas, according to the experience of authorities, it is extremely rare. Our author does not distinguish between a lobar induration, simulated by the aggregation of inflamed lobules, and that caused by exudation of fibrine—"by bleeding into the lung," as it has been expressively termed. Although we are told that in croupous pneumonia the epithelium of the alveoli is unaltered, we are nevertheless warned that the unre-

solved products of the disease may lead to phthisis. According to Buhl, this sequel is never observed except in desquamative pneumonia. The importance of lymphoid infiltration, and of lymphatic engorgement in the development of phthisis from chronic pneumonia, is overlooked by our author. Perhaps it is on this account that a separate chapter is devoted to enlargement of the bronchial glands, a lesion which, as a sequel to chronic pulmonary inflammations, deserves only to be considered in connection with them. Dr. Day gives one or two interesting histories, showing that suppuration of such glands may be mistaken for pulmonary caverns; but passes over lightly the caseous pneumonia, or even tuberculosis, which co-existed with such lesions in cases described.

It is ungracious to insist exclusively on the negative aspects of a book. But in this one there are really few positive elements requiring attention. One of the most interesting, is the record of a case of intussusception in a child of 2 years and 9 months old, treated by Dr. Day, and terminating in complete recovery.

[M. P. J.]

Anatomical Studies upon Brains of Criminals. A Contribution to Anthropology, Medicine, Jurisprudence, and Psychology. By Moriz Benedikt, Professor at Vienna. Translated from the German by E. P. Fowler, M.D., New York, Department of Translation of the Medico-Chirurgical Society. New York: Wm. Wood & Co., 1881, pp. 185.

The original of this translation was written in the summer of 1878. The author presents a description of the external conformation of 22 brains of criminals, with an analysis of the confluence of the principal fissures in 19 cases. From these facts he attempts to establish an abnormal type of cerebral fissures, which he calls the confluent-fissure type. He furthermore claims that the brains of the criminals presented belong to this type.

Reference is made in the preface to some of the noted contributors to cerebral anatomy; no mention is made, however, of one to whom we are indebted, perhaps more than to any one else, for our knowledge on this subject, namely, Prof. Theo. Meynert, of Vienna. This omission will not surprise those who are acquainted with certain local jealousies that exist in Vienna, to which vague reference is made by the author with an evident attempt to establish himself as a scientific martyr. Of the numerous interesting questions discussed by our author which are open to criticism, let us consider first the main point which he attempts to prove, namely: that there is a special type of confluent fissures, which indicates inferior

development. The author states that "the most important characteristic of this type consists in this. If we imagine the fissures to be water courses, it might be said that a body floating in any one of them could enter almost all the others." "For some time a marked fissuring of the brain was regarded, erroneously, as a sign of high development. It is true that if in the ascending scale of animal life there appears a new typical fissure, it signifies, as a rule, an extended development of the surrounding cerebral region. But when there is no new development around the fissure, and especially when the more marked fissure results from a junction of typical fissures, the fissure thus emphasized indicates a defect arising from the absence of annectants." We contend that "marked fissuring of the brain" is still regarded, and not erroneously, as a sign of high development. Increased growth gives rise to more complicated convolutions, and, necessarily, to more extensively meandering fissures; a reduction in the complexity of convolutions must, on the other hand, lead to the simplification of the fissures, and to a reduction in the number of the secondary and tertiary fissures in particular. In other words, it is a deficiency of fissure formation, not an excess, as our author states, which is a result of deficient gyrus development. We heartily agree with the author when he says that "for many of the descriptive details here given, such as are absent in all previous cerebral representations, we are indebted to the special attention which I have bestowed upon these brain specimens." This view is strengthened by an inspection of the original photographs of the brains examined; "atypy" is exhibited to a greater degree by distortions and confluent fissures due to improper treatment of the specimen, than to ante-mortem conditions. The author states, however, that "in endeavoring to describe any given brain, great numbers of details are observed which are difficult to delineate. In some brains we encounter an exhibition such as in other brains at least escapes observation. If now we revise these other brains in this respect, then this exhibition becomes here and there more or less plainly expressed," etc. It is just this revision to suit a pet theory to which we object. There is no more ample field in anatomy for a prolific imagination to advance untrammeled than just here among the cerebral convolutions, and our author has convinced us that his efforts in this direction have not fallen short of his former productions.

The second proposition stated is, that the brains of criminals presented belong to the confluent-fissure type, which leads him to

the conclusion that "the brains of criminals exhibit a deviation from the normal type; and criminals are to be viewed as an anthropological variety of their species, at least among the cultured races." When we consider the small number of brains examined, the admission that we have as yet had no comparative race-study of brains, and his statement that the larger proportion of brains found in dissecting-rooms belongs to the confluent-fissure type, we are not prepared to dispute the author's own statement that "it is self-evident that the observations here collected are the result of an *a priori* conviction," etc. He wisely adds, that the matter should not leave the hands of the anatomist without further proof.

We must dissent from the author's sweeping assertion that "there exists no qualitative difference between the brains of mammalia and those of primates," in the sense which he applies it to the cerebral convolutions. The search after homologies is one that requires extreme care. That numerous homologies can be traced between the brains of lower mammals and those of the primates, is undoubted; but there is a limit beyond which this process cannot be carried with certainty. The general facts of evolution teach us caution in this respect. The same law must apply here which applies to other organs. The effects of Transmission, which tends to reproduce in progeny the characteristics of progenitors, are modified by Adaptation to varying circumstances, which produces continual variation; these two tendencies produce constantly diverging forms; so that when the phylogenic relationship between two animals is remote, the chances of tracing homologies become proportionately less, and when found, present indefinite characteristics. The statement, then, that "architecturally there exists no fissure arrangement (idea) in the animal brain which has not been expressed in the human," cannot be true, for we could not include morphological variations in the "animal" brain which arose after a divergence from the common type had taken place. It is not a series with which we have to do, but divergencies.

The laudable efforts to put the study of crime on a more scientific basis cannot be too highly praised; we must be on our guard, however, against views concerning so manifold a subject, when they are based on a small number of observations of doubtful import, though they be supported by *a priori* argument. A plausible theory may lead us to the discovery and interpretation of important facts; or it may, on the contrary, force us to distort and

mask the truth. We regard the conclusions of the author as premature ; too little is known as yet concerning the limits of the typical topography of the cerebral convolutions to class as atypical conditions which the author claims are found in dissecting-room subjects in the larger proportion of cases ; but if the work leads to a more extensive and thorough comparative study of the brain in different races, it will not have been written in vain.

The wood-cuts, which the translator considers compare favorably with the beautiful photographs of the original, are coarse and the impressions poor ; otherwise the book presents a neat appearance. [W. R. B.]

ORIGINAL OBSERVATIONS.

TUMOR OF THE PONS VAROLII.

By F. T. MILES, M. D.,

PROFESSOR OF PHYSIOLOGY, AND CLINICAL PROFESSOR OF NERVOUS DISEASES, UNIVERSITY OF MARYLAND; MEMBER OF THE AMERICAN NEUROLOGICAL ASSOCIATION.

Florence Brown, æt. 17, well developed, was brought into the University Hospital in a stupid, apathetic condition, which made it difficult to obtain from her her history.

She said she had been sick for more than a week before coming to the hospital, that she had had a fall, after which she gradually grew worse, but that she had been sick before the fall. She presented the symptoms, well-marked, of a crossed paralysis, the left side of the face and the right arm and leg being affected. The paralysis of the left side of the face was complete, so that she could not close the left eye nor corrugate the brow on that side. The right arm was completely paralyzed, the right leg partially. She could walk feebly, dragging the right foot. No incoördination was observed. Sensation, tested for in various ways, appeared to be completely abolished in the left side of the face, including the conjunctiva. Ammonia held to the left nostril, the right being closed, caused no irritation. The tongue could be protruded, but the sensitiveness of the two sides could not be conclusively tested on account of the hebitude of the patient. Sensation was very greatly impaired, if not totally lost in the right hand and arm, and decidedly diminished in the right foot and leg. The sensitiveness of the trunk was not tested. The hearing of the left ear was blunted. The globe of the left eye remained fixed and immovable. There was no strabismus. The conjunctiva was inflamed, and the cornea so cloudy from recent inflammation that the pupil could not be observed. Deglutition was somewhat

interfered with, but she ate with appetite. There was no vomiting. The temperature varied within narrow limits, being sometimes above and sometimes below the normal. Pulse, frequent and weak.

She passed her evacuations in bed, but this appeared rather the result of her apathetic condition than of a paralysis of sphincter or bladder. Indeed, her mental faculties were so much blunted, and it was so difficult to rouse and fix her attention, that it was almost impossible to thoroughly investigate the symptoms.

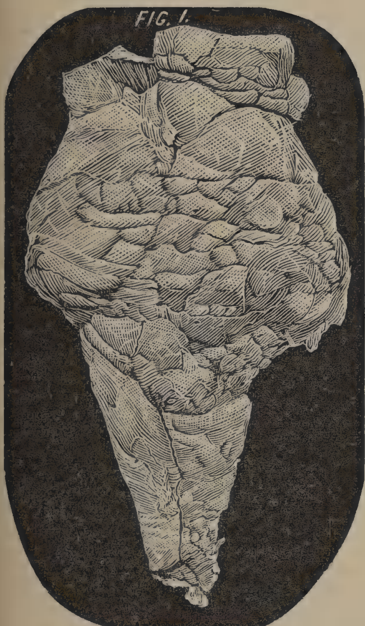


FIG. 1.—Nodulated growth on anterior surface of pons.

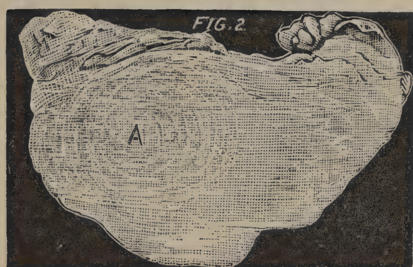


FIG. 2.—Transverse section of pons, showing the location of the intra-pontine growth.

A diagnosis was made of a lesion in the region of the left half of the pons, with probably a thickening of the dura mater of syphilitic origin. She was prescribed active antisyphilitic treatment, but without effect. She sank rapidly, dying comatose, without convulsions.

The autopsy showed the membranes healthy in appearance, the hemispheres and cerebellum normal throughout. The pons was remarkedly distorted, and apparently hypertrophied, as shown in the wood-cut taken from a photograph of the specimen. Not only was it unsymmetrical, owing to an enlargement of the left

half, but its surface was uneven and nodulated, the upper and lower margins projecting over the cerebral peduncles and medulla. The peduncles, particularly the left, showed somewhat of this swollen and nodulated character. The anterior pyramids were much distorted, the right pushed aside, the left deeply indented and as if compressed by the swollen margin of the pons. The floor of the fourth ventricle was distorted on the left of the median fissure, being widened and bulged upward. After hardening in alcohol, a transverse section was made through the pons a little nearer its lower than its upper margin, passing through the middle peduncle of the cerebellum, the face of which section is shown in fig. 2. There appeared a growth occupying the left half of the pons, and encroaching on, or rather pushing aside the raphe. Although it merged into the surrounding tissue, which it had apparently pushed aside, without contrast of color, or distinct line of demarcation, yet the circular shape of its section was shown by faint concentric markings here and there throughout its substance. Other sections proved the mass to be nearly spherical in shape. Near its centre there was an appearance of slight disintegration.

The symptoms in this case were for the most part typical of unilateral lesion of the pons Varolii; almost all of them find their explanation in the position of the new growth. The motor (pyramidal) tracts and the tracts of sensation which connect the limbs of the right side with the left hemisphere, were interrupted by the tumor in the pons above their decussation in the medulla, while we may suppose that the nuclei of the fifth, sixth, and portio dura were involved in the growth, causing paralysis of those nerves on the left side, and thus giving the crossed, or alternate paralysis so often seen in pons lesion. The complete paralysis of the facial, resulting from the implication of its nucleus, is distinctive of lesion of the pons, as compared to its partial paralysis from lesion of the hemisphere or its ganglia. With such an extensive lesion it is difficult to see how the tracts of sensation and volition for the nerves of the right side of the face escaped, and yet that they did so would appear from the complete absence of anæsthesia and paralysis on that side.

Paralysis of the oculo-motor nerve, such as existed in this case, would not be expected in a lesion of the pons, the nucleus of that nerve being situated too high up. Nothnagel says[1] that a paralysis of the bulbo-muscular branches of this nerve proves an

[1] Topische Diagnostik der Gehirnkrankheiten. Berlin, 1879.

extension of the disease beyond the pons, except, perhaps, in cases of conjugate deviation of the eyes. Likewise with regard to the inflammation of the conjunctiva and cornea: the same author treating of lesions of the pons Varolii says:[1] "Vasomotor lesions of the face are not described; the 'neuroparalytic' inflammation of the bulb accompanying lesion of the trigeminus belongs certainly *not* to the symptoms of pons lesion, according to the cases at present recorded. In short, they are absent in almost all intra-pontine lesions of the trigeminus." There was no appearance of alteration of the nerve after its exit from the pons, nor of the ganglion of Gasser. It is possible from the remarkable hypertrophic and nodulated character of the surface of the pons, that pressure may have been exerted upon the nerve trunk sufficient to produce the trophic disturbances observed. The same may be said of the trunk of the oculo-motor, the rather as we see the swollen and nodulated condition referred to, extending upon the surface of the left crus cerebri. We would thus have, joined to the symptoms of simple intra-pontine lesion, those which were the result of compression of nerve trunks. The specimen has not yet been examined microscopically.

A CASE OF MYSOPHOBIA.

By J. C. SHAW, M.D., Brooklyn.

E. J., male, aged 15 years, brought to me by his mother on May 26, 1879.

For the past few years has enjoyed good health. About six weeks ago the first decided symptoms appeared, but for months before, his mother had noticed that he was excessively particular to wash his hands very clean, which is unusual for boys, as she remarks. About six weeks ago he began to say to her: "He had been touching the paint; did she think it could come off the wall and poison him?" He would not take off his own hat, but ask her to do it for him; also, to unbutton his coat. If he wished to come in the front door he would not take out his night-key and come in, but would knock on the door with his elbow. He is afraid that if he touches any thing with his hands it will poison him; every time he touches *any thing* he is very particular to wash his

[1] *Ibid.*

hands very clean. He spat on the carpet a few days ago, and then rubbed it off with his boot; he immediately came down stairs to his mother, told her about it, and asked if she thought he could have got any of the color off the carpet so as to poison him. He would go about holding his hands and arms away from his body, as if he were afraid of touching his clothes. When he goes to bed at night he will wash his hands a dozen times and use as many towels; if prevented from doing this he appears disturbed, and will sometimes rush over to the water-pitcher and thrust his hands in, which appears to satisfy him. For a short time past he has frequently asked his mother if he had cobwebs on his face, and especially about his mouth. His mother thinks that of late he has presented a vacant, idiotic expression that he never used to have.

When the boy is talked to he speaks sensibly, but will give no explanation of why he is afraid of being poisoned by touching things; says he has frontal headache at times, especially when he goes to school; for the past six weeks has not gone to school, and has not had the headache, but a few days ago had a sharp pain in left occipital region. He looks dull and apathetic; is not given to self-abuse. He has a decided neurotic family history: the brother and father of his mother suffer from some nervous disorder, but no accurate account of it could be obtained; but I infer that it is a mental disorder. The marked neurotic family history in this case inclines me to the opinion that graver mental disorder will be developed in this boy.

Mysophobia was first described by Dr. Wm. A. Hammond in 1879,[1] a few months previous to my seeing the above case.

Dr. E. C. Seguin has since reported a case.[2]

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[1] *Neurological Contributions*, vol. i, p. 40.

[2] ARCHIVES OF MEDICINE, August, 1880.

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ARCHIVES OF MEDICINE.

Original Articles.

A CLINICAL STUDY OF LEPROSY.*

By HENRY DICKSON BRUNS, M.D.,
NEW ORLEANS.

THE proneness of the older authorities on diseases of the skin to excessive subdivision, long aided in hindering a proper appreciation of this subject.

Happily the increase of more exact pathological knowledge has greatly simplified our ideas upon this class of diseases, and in considering the pathology of leprosy I shall endeavor to show that the so-called varieties are dependent upon what might be termed a pathological accident.

Proceeding to the examination of the clinical grounds for the various divisions of the disease into varieties, the older and more minute subdivisions are passed over, only such as are found in recent works being considered.

Most modern authorities divide the disease into the tubercular and anæsthetic varieties; a third variety, the macular, is added by some.

Even those, however, who divide the disease into these two distinct varieties, are forced to admit that they may, nay, very often do, succeed one upon the other. Thus, Danielssen and Boëck state that the anæsthetic complicates

* Extract from a prize thesis of that title submitted to the Faculty of Jefferson Medical College, March, 1881, and now printed by their kind permission.

the tubercular variety in one in every six cases, while in one case in every twenty the anæsthetic variety becomes tubercular. Hansen, however, not admitting an anæsthetic variety, holds that anæsthesia occurs, not as a mere chance complication, but regularly late in the course of every tubercular case, provided the patient survives long enough to allow of its manifesting itself. He demonstrated the presence of anæsthesia in 135 out of 144 tubercular cases.[1] The nine in which it was absent were all of recent origin. (*Archiv für Dermat. u. Syph.*, 1871. Kaposi, *loc. cit.*) Hansen's opinion is supported by Bidenkap (*Norsk Mag. for Laege*. v. iv. Kaposi, *loc. cit.*) Dr. J. H. Bemiss, writing from the Sandwich Islands, says: "There are present here the two forms: tubercular and anæsthetic. This division is based upon the predominance of one or the other of the two most important symptoms. In actual observation the disease does not always admit of such strict classification, but generally shows the two forms combined in greater or less pathologic preponderance. It may start as purely tubercular or purely anæsthetic, but does not often preserve a single type throughout its course; in the one case anæsthesia, in the other tubercles, making their appearance in due time."

My own experience coincides exactly with that of Dr. Bemiss. I have never examined a case of tubercular leprosy in which more or less anæsthesia could not be demonstrated. By reference to the analysis of cases, it will be seen that out of fifteen tubercular cases twelve were also anæsthetic. Of the two anæsthetic cases given in the table, one developed tubercles within three years from the first

[1] It will appear further on that anæsthesia may be due to two causes: pressure upon the nerve trunks (the lesion in the true anæsthetic type), and pressure upon the peripheral nerves (a phenomenon which *undoubtedly* occurs in all old-standing tubercular cases). To which of these causes the anæsthesia in Hansen's cases was due does not appear. Anæsthesia should always be determined by thrusting (as apparently was done here) a needle deeply into the true skin; otherwise we may be deceived by thickened epidermis.

attack; the other now presents infiltration of the brow and eyelids, one of the initial symptoms of tubercular leprosy.

With Drs. Kaposi and Bemiss, then, I conclude that the varieties (as now made) are dependent on symptoms which are by no means constant, but vary with varying causes.[1] But in the following description I shall classify as tubercular in type, preferring the word used by Dr. Kaposi, all those cases in which the symptoms are mainly due to the presence of leprous new-formation in the corium and mucous membrane, and as anæsthetic,[2] those cases alone which owe their striking features to compression of the nerve *trunks* by the same neoplasm; for thereby the study of the disease will be much facilitated, so widely do the two groups differ in their clinical manifestations. At the same time, I repeat that the division is, on the whole, an arbitrary one, and that the two types may, and as a rule do, complicate one another in old-standing cases.

"The macular variety" is a phrase employed with varying signification by various writers. Some place under this title those cases which manifest the maculæ characteristic of an early stage of the tubercular type, in which tubercles are as yet absent. This, however, as I have just said, is not a variety, but merely one stage of one type of the disease. Indeed, I am inclined to believe it somewhat uncommon, at least in Louisiana, to see this stage clearly defined, *i. e.*, the maculæ existing without tubercles or infiltration. I have never met with such a case, and Dr. J. H. Bemiss remarks: "As to a third variety, macular, my observation does not warrant my stating any such class. True, maculæ of one sort or another are common enough, but these cases already present one or the other of the two forms generally recog-

[1] The disease undoubtedly presents also slight variations in form in different localities, a fact which may serve to explain, in part, the nonconformity of the descriptions given by authors who have studied the disease in different countries.

[2] *I. e.*, in type.

nized." The doctor then describes two cases in which maculæ were the most prominent symptom, but in one a few tubercles had already made their appearance, and in the other infiltration around the alæ nasi was beginning. This is a point which is, I think, not sufficiently dwelt upon. Many of the descriptions would lead one unacquainted with the disease to suppose that cases presenting numerous and conspicuous maculæ were exceedingly common.

There are other authorities who regard morphœa as a macular variety of leprosy. Thus, Erasmus Wilson speaks of it as an impression of the gigantic footsteps of that grand, that elephant disease, the leprosy of the middle ages. But the infiltration, the varying degrees of anæsthesia, the loss of hair from the affected part, although present in leprosy, are not primary, but secondary symptoms. They may be the result of any infiltration which seriously interferes with the circulation through the skin, and to disturbances of function in the trophic and sensory nerves. The infiltration, too, of morphœa differs in character from that of leprosy. Again, the purely local and comparatively trivial nature of morphœa does not, I think, warrant for it the supposition of a constitutional cause. True, it occurs principally in females and debilitated individuals (Tilbury Fox), but this is a strong argument against a specific cause. For an inherently weak spot in the organism may not, so long as the general health remains unimpaired, present any untoward appearance, but, should the nutrition of the whole body sink to a low ebb, it must of necessity suffer more gravely than any other part.

For these reasons, therefore, I do not believe in the identity of morphœa and leprosy.

The symptoms of leprosy may be divided into general and local.

The general symptoms are common to both types, and are by no means characteristic. I shall consider them first.

With whatever form of the disease the patient may be afflicted, if it has endured for any considerable length of time, he will always present a miserably emaciated appearance. The fat has in great part disappeared from the body, and the muscles are soft and atrophied. The skin usually participates in this emaciation, and has a thin, finely-wrinkled appearance like tissue-paper. I have specially noted this on the hands.

In the atrophying muscles of both types, but especially in the anæsthetic, local contractions, or twitchings of a few muscular fibres here and there, are to be observed. If not present at the time of inspection, they may frequently be excited by smartly tapping the muscle with the finger. Such contractions Dr. Hammond terms fibrillar in his work on diseases of the nervous system.

They are common in many nervous diseases accompanied by wasting of the muscles. In the muscles composing the ball of the thumb, and in those around the mouth and orbit, they are of most frequent occurrence.

Œdema, as in many states of great depression and disturbance of circulation, is common, and is most marked in the lower extremities.

Swelling of the inguinal glands is not unusual, especially in the tubercular type. I have not observed induration of any other glands, but it is said occasionally to occur.

Turning to the appended charts it will be seen that the temperature of the three male patients rarely sank below 99° F., the averages being all above this degree, and that there was a slight but constant evening elevation. The charts of the two females, presenting almost normal curves, are of little interest, save that one of them (Deneina Boyens) serves to illustrate, more markedly even than any of the male patients' charts, the sudden, high, and brief febrile paroxysms to which lepers are subject.

It is said that the temperature of the surface of anæsthetic parts is appreciably lower than the general temperature of the body. This is what might have been supposed *a priori*, but I have not had the means of verifying it.

The pulse in the three male patients just alluded to was fast and weak, the averages, taken in the sitting posture, being about 102, 85, and 89 beats per minute. There was a slight falling off in the number of beats per minute, toward evening. This rapid and weak pulse appears to be a characteristic of the disease, for Dr. J. Kinnis states the average pulse rate (sitting posture) at 100–110 beats per minute: weak. The most rapid pulse which he encountered was 124, the slowest 88 beats per minute.

Surgeon Major W. I. Van Someren gives the following figures: Out of 426 cases the pulse was below 70 beats per minute in 57 cases, from 70 to 80 in 100 cases, from 80 to 90 in 132 cases, from 90 to 100 in 83 cases, 100 beats and upward per minute in 54 cases.

It has been stated that the pulse of the anæsthetic type is habitually slow, but in the case of this type under my charge the average morning pulse was 90$\frac{5}{7}$ beats per minute, the evening, 87$\frac{1}{2}$ (Antoine Gaspard).

Albuminous urine seems to be regarded as a characteristic symptom by most authors, but in none of my cases was it present (see charts). In a number of examinations made by Van Someren, the urine was found to be more or less albuminous in 40 out of 100 cases, saccharine in 1, phosphatic in 37, alkaline in 42, acid in 2, and neutral in 52.

At various epochs the unfortunate leper has been either an object of abhorrence and disgust on account of a suppositious salacity, or of contempt and pity as the victim of sterility and impotence. The former idea is utterly, the latter, partially false: that is to say, leprosy does not of necessity produce sterility, although the leper may be reduced

to such a state either as the result of the depression of general health[1] under which he labors, or by reason of the testicle becoming involved in the morbid process.

Lastly, before passing to the consideration of the local symptoms, mention should be made of the overpowering languor, hebetude, and drowsiness which invariably possess the leprous patient.

In the description of the local symptoms, I shall take up first the tubercular, and then the anæsthetic type. For purposes of clinical convenience, each type may be said to have three stages; this, however, is an arbitrary division: one stage glides into another without any sharp line of demarcation, maculæ and tubercles coexisting, as a rule.

TUBERCULAR TYPE—morbid changes chiefly in corium—may be either acute or chronic. Divisible into:

First, or prodromic stage;
Second, or macular stage;
Third, or tubercular stage.

Chronic form: Prodromic stage.

Some months before the appearance of any objective symptoms, the leprous subject usually experiences those disturbances of general health which, almost always, precede a severe attack of illness. He suffers from lassitude, malaise, loss of appetite, nausea, epigastric oppression, indigestion, slight fever with evening exacerbations, or paroxysms of chills and fever. A chronic bullous eruption, lasting days or months, and strongly resembling that of pemphigus, is also described as one of the premonitory symptoms. This eruption was formerly maintained to be pathognomonic of the anæsthetic type, but it is impossible to foretell the appearance of a certain type by means of this or any known prodromic symptom.

In two of my cases, epistaxis was a promonitory symp-

[1] Dr. Enders asserts that leprous women are generally barren.

tom. Dr Kinnis, also, has noted this and brief febrile attacks in the earlier stages of leprosy in Ceylon. Only 5 out of 17 of my cases are set down in the analysis as having had any prodromic stage, and out of 117 cases (examined by Van Someren) which had a history of antecedent "malarial fever," 52 had no other prodromes. Doubtless all of these figures fall far short of the truth, but those who have had most experience will best appreciate the difficulty of obtaining an intelligent "previous history" from the class which frequents large hospitals.

After these prodromes have lasted for months or years (they may have been entirely absent), the second stage is ushered in by the appearance of the first maculæ.

Second—Macular stage.

As the maculæ characteristic of this stage make their appearance, the prodromic symptoms just described, in most cases, disappear.

These maculæ are claret-colored, the color vanishing on pressure, and slightly elevated, their outlines being either clearly or ill-defined. In size they vary from the palm of the hand to a finger-nail, but are usually about the dimension of a silver dollar. Their favorite sites are the trunk and the extensor surfaces of the upper and lower extremities. They are occasionally seen on the face. On touching one of these spots, the skin is perceived to be harsh, infiltrated, and hyperæsthetic; rarely normal as to sensibility. Gradually, however, the original and ruddy hue of the macula becomes a light coffee-color, which does not disappear under pressure, and hyperæsthesia gives place to anæsthesia.

Now the cuticle commences to desquamate lightly, and the patches look dry, tense, and shining, or unctuous from hypersecretion of sebaceous matter. The latter condition is more frequently seen in negroes.

Among them also the spots are said to be reddish or copper-colored (Campet, *l. c.*); in the yellow races they may be of lighter or darker shade than the normal skin. After a certain time the macula disappears completely, or atrophy of the skin takes place; a shining, brilliant white, finely wrinkled spot, destitute of glands and hair, remaining. Occasionally tubercles crop out upon the former sites of the maculæ. The latter continue thus to appear and disappear at intervals of a few weeks, or several months (half a year); slight febrile symptoms preceding, as a rule, the advent of each fresh crop.

The later crops, however, remain as a permanent brown discoloration of the skin which gradually spreads and deepens until it involves the whole surface; the shade being darkest upon those portions of the person habitually exposed to the air.

An erythematous blush diffused over a considerable area and preceded, or not, by maculæ, is by no means a rare precursor of tubercular deposition.

As this blush fades away, the skin acquires the characteristic brownish tint, and in a short time tubercles are deposited. Dr. J. H. Bemiss describes such a case: a Sandwich Island native came to him "with the left side of the face swollen, painful, and presenting all the characteristics of phlegmonous erysipelas, for which he was treated." Finally the pain and redness passed off, leaving a hypertrophied state of the skin. Here the tubercles were subsequently deposited, not at the seat of the former inflammation, but "upon the pharynx and posterior part of the tongue." At the same time the right hand became partially anæsthetic. Twice only have young maculæ come under my observation. On both occasions they were claret-colored, and hyperæsthetic. Tubercles and infiltration were also present. The macular stage may, however, precede the tubercular by

as much as five years (Danielssen and Boëck). In a case mentioned by Erasmus Wilson maculæ and discoloration were the only symptoms for five or six years; at the end of this period tubercles appeared.

Third.—Tubercular stage.

Over the whole body, especially over the face and lower parts of the extremities, the skin has now assumed a dark appearance. It is hard to convey in words an exact idea of this tint. On the trunk and upper parts of the limbs the skin is about the color of a mulatto's, or the color of the light brownish-yellow blotches seen upon the persons of pregnant women, or, more exactly, of the dark patches of leucoderma. Upon the exposed portions of the person the shade may be the same, but usually deepens to a dusky reddish-brown. The "Atlas of Skin Diseases" by Dr. Tilbury Fox contains a plate in which the color is well represented, but the shade is much darker than I have ever seen it.

Upon the parts first attacked, as a rule, face, hands, or feet, the skin is usually found thickened, and looking semi-translucent, as though a gelatinous material lay immediately beneath a discolored cuticle, especially around the alæ nasi, over the malar bones, and in the lobes of the ear.

There may be, at this early period of the stage, however, no discoloration, a delicate pink blush, with here and there a fine tortuous venule, overspreading the thickened skin.

The eyebrows, lashes, and beard have now fallen, or become scant, the lobes of the ear still more pendulous, and the alæ nasi flat and spreading.

Large thick plates of an elastic-feeling substance are then deposited in these localities. As tubercles begin to appear in well-marked crops, the erythematous condition and semi-transparent look of the skin subside, as a rule, but I have

known them to coexist with tubercles for a considerable time.

The early prodromic symptoms of lassitude, fever, nocturnal pains, etc., often precede the advent of a crop of tubercles, and then pass away after the critical period, leaving the patient much more comfortable.

Tubercles are most common on the face in my experience; then on the extensor surface of the upper and lower extremities. I have never met with them on the trunk, although they may occur in this situation.

On the hands they cluster thickly upon the extensor side of the phalangeal joints, interfering seriously with motion. Sometimes they affect the toes in like manner, seeming prone, in fact, to collect around any and all joints. Tubercles may be solitary and scattered, but in their chosen localities are usually found matted together in nodular clumps some inches in extent.

Such groups are commonly seen in the skin of the brows, deepening the natural wrinkles into furrows, over the triceps extensor cubiti, and on the back of the hand. In shape these growths are hemispherical, or slightly conical, with a base broad in comparison to the height. They vary from the size of a pea to that of a chestnut. Such small nodules as the first mentioned have been observed clustered in groups and circles like the deposits in lupus.

On handling one of these bodies it is perceived to be embedded in the corium and the subcutaneous connective tissue, and usually may be made to slide freely over the subjacent bone or muscle. Tubercles are firm, elastic, painful upon pressure; often preserving the previously described translucent appearance when it has wholly vanished elsewhere. The skin over the node is thin, smooth, shining, with lightly desquamating cuticle.

At this period the small superficial blood-vessels once be-

fore alluded to are fewer, but of larger size, red and tortuous. They radiate toward a centre, where they dip down into the skin, and are lost to sight, recalling exactly the retinal vessels seen with the ophthalmoscope. They are most numerous by far upon the face, but may also be found upon the chest and hands.

Flat or nodulated masses are identical in all these respects with the tubercles.

After a varying length of time the morbid process next attacks the mucous membranes. Tubercles exactly resembling those in the skin, though usually of smaller size, are deposited in the membranes of the nose, the mouth, the pharynx, the larynx, the bronchial tubes, the intestines, and the eye, in the order given.

All visible portions of these mucous surfaces become thickened, and deeply covered with an ashen-gray epithelium in the greater part of their extent. Thus the interior of the nostrils is pale, or grayish, with here and there dry, red fissures, or small tubercles. The tongue is coated down the centre with a broad, grayish-yellow stripe, leaving only the edges and tip of a pale, unhealthy-looking pink. The papillæ are prominent ; the surface marked with small excoriations, occasionally studded with tubercles, causing a curious lumpy appearance and some loss of mobility. Similar characteristics are presented by the membrane of mouth, pharynx, and larynx. The hard palate, it is said, is usually covered by a flat plate of infiltration, which is sometimes dotted over with tubercles, single or in groups, showing red upon the dull gray ground.

It is hardly necessary to state that the voice is greatly altered, or may even be completely destroyed by these changes, and that occurring in the larynx they may endanger life.

When the eye is attacked, the first phenomenon observed

is general and intense injection of the conjunctiva, accompanied by lacrymation.

Some time after this a more or less extensive pannus (pannus leprosus) may be observed encroaching upon the edge and spreading over the surface of the cornea. The process may then pause here, as I have seen it do, the injection disappearing, and the pannus seeming to become much thinner and more transparent, from the emptying of the formerly distended capillaries. At this stage close inspection will make out, as the result of the previous inflammation, some thickening, and a few minute tubercles not larger than grains of sand, around the cornea. If, instead of ceasing at this point, the morbid action, on the contrary, continues, the cornea may be perforated, synechia anterior resulting; or, deposits which have now been thrown out upon the iris, extend across the posterior chamber to the lens, synechia posterior (Dr. Kaposi, *loc. cit.*) Then the eye may be destroyed by atrophy or softening. At times an acute general ophthalmia sets in and hastens the total destruction. Great pain may accompany these changes.

And now we reach the "last scene of all that ends this strange eventful history,"—retrogression of the neoplasm, and all its horrible consequences. Those crops of tubercles, and the same is true for all forms of deposit which first appear, are, as a rule, soon absorbed, but only to be succeeded by larger and more stable crops, individual members of which may persist for years.

Three forms of retrograde metamorphosis are enumerated.

I cannot, however, regard as material the difference between the second and third forms.

1. Atrophy: the tubercle undergoes atrophy or absorption from the apex toward the base. The cuticle desquamates the while, and the whole mass disappears in

days or weeks, a thin, contracted, wrinkled, light brown, or white spot of atrophied skin remaining. Young tubercles, it is said, are apt to be deposited around this spot.

2. Softening: the tubercle softens or breaks down at the top, pouring out a cheesy, purulent discharge through one or several openings. The base remains, or disappears by atrophy and absorption.

3. Ulceration: due, according to most authorities, to accidental mechanical causes.

Mechanical or therapeutical irritation, and pus pent under infiltrated masses, are ascribed as causes of erysipelas or lymphangitis during this process.

The ulcers produced are worthy of further consideration. They are sluggish, non-granulating, marked by great necrosis of tissue.

Occasionally of small size, with flat edges, and presenting the smooth, yellowish pink surface common to non-granulating sores, they are usually, in my experience, large, with high, perpendicular, hard edges.

These edges are either of a white or faint pink hue, or of the bluish tint peculiar to young scars.

The base of the ulcer has a fleecy look, not unlike the woolly side of a piece of patent lint. This is due to fine fibrillæ of tissue which, having to some extent resisted the ulcerative process, project beyond the general level.

As the ulcerated surface is of a dark red color, finely dotted and streaked with various shades of yellow, dark blue, olive-green, etc., the impression produced is of a yellowish-red surface marbled in very dark blue, or green. I have frequently seen small, dark yellow masses of dead tissue projecting from the base of an ulcer, but the fungous growths described by some have never come under my observation.

At times these ulcers attain enormous dimensions, cover-

ing and encircling a foot and leg; situations in which I have always observed the larger ones. In such a case the discharge is thin and ichorous, and the ulcers manifest no tendency to heal. As in all chronic inflammations, the surrounding structures are thickened and discolored.

When the smaller ulcers heal the scars produced are thin and pale. Strong contraction takes place in the cicatrix, producing an appearance of radiation from a central elevated point, which is heightened by the distribution of a dark coloring matter along the lines of radiation through the light colored-scar.

All, or almost all, of the horrible and repulsive features which have made the name of leprosy a terror for ages, and the miserable leper an outcast—anathema—upon the face of the earth, are due to the ulcerative process just described.

On the face tubercles rarely break down; but I have seen, as the result of such an occurrence, a patient with a small, cup-like, running sore upon the cheek.

The nasal cartilages are early, but the bones are not, attacked as in syphilis. Distortion of the organ results, for the bones remain prominent, while the tip becomes broad, thick, flattened, and turned up.

Around and in the eye ectropion and ulceration of the cornea are the results of these changes. In the mouth they may cause loss of the uvula, and produce small sores which give rise to pain on swallowing warm food. One of my patients complained constantly of this. Great fetor of the breath may accompany the breaking-down of deposits in the mouth.

It is, however, upon the hands and feet that we perceive the saddest effects of ulceration. When this occurs around the nails they scale off, leaving the matrix exposed and raw. Should an ulcer form over a phalangeal joint it becomes

gangrenous, deepens rapidly, destroys and removes the distal part of the finger or toe. Frequently softening with discharge, or absorption of the bone and other tissues of an intermediate phalanx, takes place, and the parts retracting, the third, is drawn down upon the first phalanx. Upon hands thus deformed, the fingers stand out at every conceivable angle, it is said, producing a most curious effect. Fortunately, little or no pain accompanies this mutilation, and the healing process is very rapid.

Tubercular type—Acute form.

Most authors also describe an acute form of this type, which differs, not in the nature of the morbid processes, but in the degree of rapidity with which they run their course. Its prodromic and eruptive stages are characterized by considerable fever, and the tubercles instead of appearing and disappearing in crops, burst forth once and for all in great numbers. In a fortnight ravages are committed which it would take the chronic form months to inflict. After a certain time this form may become chronic, and in that case the disease runs its usual course: should it, on the other hand, continue acute, cerebral complications, pneumonia, pleurisy, chills with febrile exacerbations, within a few months close the scene. Occasionally the fever may be typhoid in character, but usually presents the common symptoms of high temperature, rapid pulse (120–130 per minute), thirst, insomnia, headache, delirium, constipation, and high-colored urine. The initial attack of leprosy is rarely made in the acute form, but chronic cases frequently terminate in this manner.

Anæsthetic type—Morbid process chiefly in sheaths of nerve trunks.

Form, invariably chronic.

The anæsthetic type, while almost unknown in certain latitudes, seems in others to be the prevailing form of

leprosy.[1] The former is the case in Louisiana, I must conclude, as no report of an anæsthetic case has reached me, and only one has fallen under my observation. Indeed, it may be asserted that, on the whole, the anæsthetic is the rarer of the two types.

This type manifests itself in two distinct modes: it either supervenes upon, and complicates a tubercular case, or it exists as a distinct type from the beginning. In either case the symptoms are much the same.

Prodromic or macular symptoms precede the disease in some instances, in others it developes under the skin which in no wise indicates the presence of its chief symptom,—derangement of sensation.

This type may also, as a matter of convenience, be divided into stages; but the remarks already made concerning a like arrangement of the tubercular type, are applicable here.

First.—Prodromic stage.

There is no essential difference between this and the corresponding stage of the tubercular type.

One phenomenon alone needs more extended consideration, viz., the bullous eruption before mentioned; a symptom usually, but not always, premonitory of this type.

Occasionally preceded by a febrile movement which ceases as they come out, at other times appearing suddenly upon a raised and reddened surface, these bullæ exactly resemble those of pemphigus vulgus, but not more than two or three are present at one time. Their size varies from that of a small pea to that of an egg. After lasting hours or days the bullæ break, pouring out a clear, sanious, or "bluish-stained"[2] serum, and dry, a pigmented, or white and glistening spot remains. At other times a shallow,

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[1] Pruner, quoted by Kaposi. Egypt, New Zealand, Thomson (*loc. cit.*), Van Someren (*loc. cit.*).

[2] See Hyde, *American Practitioner* (*loc. cit.*).

sluggish ulcer is left. The ulcer heals slowly, covered by a thin scab; the resulting cicatrix being thin, devoid of hair, or covered by a fine white down. At times the bullæ do not retain the size and form in which they first appeared.

The bleb extends, forming a vesicular ring, which becomes dry and flat in centre. The spread of the inflammation in this manner may choke the central bit of tissue, causing it to become gangrenous, an unhealthy chronic ulcer resulting. Such white and atrophied spots as are formed in consequence of these changes preserve their normal sensibility for a time, but subsequently become anæsthetic. This eruption may appear years before any other symptoms manifest themselves, or, on the other hand, may crop out upon portions of skin which have lost their sensibility.

During this period it is usual for some of the other prodromic symptoms, mentioned while treating of the tubercular type, to be more or less prominent.

Second.—Macular stage.

It is rare for this stage to be clearly marked. As we have already seen, certain maculæ exist during the presence of the pemphigus-like eruption. Other maculæ, consisting of light-red spots, similar to those described under this division of the tubercular type, or various pigmented, discolored, well- or ill-defined patches are found scattered over limbs, face, or trunk.

Third.—Stage of anæsthesia and atrophy.

Some little time after the advent of the disease, and while the above-mentioned maculæ are appearing and disappearing, it is usual for hyperæsthesia to manifest itself in certain localities. The hyperæsthesia may vary in degree and extent. Sometimes it is only the white or otherwise tinted spots left by the bullous eruption which display this condition; or any of the maculæ, especially the red ones, may be hyperæsthetic when they first appear. Again, this

state may exist in the skin of certain localities only, or over the whole cutaneous surface. Finally, an erythematous blush may, after the appearance of hyperæsthesia, suffuse the affected area. In degree, the exaltation of sensibility may vary from a feeling of tingling or formication to that of acute pain, local or general in extent; excited by contact alone, or spontaneous and persistent. When the latter condition is present, the patient may start frequently during sleep, or be subject to attacks of trembling, reflex in their nature, which shake him as would the paroxysm of an ague fit. This state of course renders him perfectly helpless, so that his attendants are even obliged to feed him. During this time various nerves may be felt very much swollen and extremely sensitive. I am ignorant how long this condition may endure, but the ordinary symptoms of pricking and formications may last, with intermissions, for years before they pass into anæsthesia.

Slowly, very slowly, those spots and stains which first became hyperæsthetic descend in the scale, lose first their morbidly exalted, then their normal function, which thus passes finally on into complete extinction—anæsthesia.

Thus early in the progress of the disease the white atrophied patches of skin become anæsthetic, and this condition usually co-exists with hyperæsthesia in other localities. At this time also portions of skin entirely normal in appearance, but quite devoid of tactile sense, are frequently met with; and this anæsthesia, it is said, may have a shifting character.

Following, however, the progress of the hyperæsthesia, sensibility gradually fades from broader and broader areas, and from deeper- and yet deeper-lying structures, so that skin, cellular tissue, and muscle become at last little more than "senseless things." As a rule, this process is confined to certain regions, an extremity or a circumscribed extent

of surface, which may be points of distribution for given sensory nerves. But this is by no means always the case, for the anæsthesia may invade, without entirely occupying the area controlled by several distinct sensory filaments; islands of normal skin being found here and there.

Subsequently, as the disease progresses, the anæsthesia creeps insidiously on, until the whole body may become involved, and only scattered points remain normal.[1]

With the loss of sensibility other changes have been keeping pace. The skin over the anæsthetic regions has become thin, finely-wrinkled, harsh, livid, or of a light-brown hue.

Its functions are destroyed, and temperature lowered.

The hair falls, especially upon the face. Where it remains upon anæsthetic spots, it may turn white, and it is asserted that the hair over the entire cutaneous surface may be blanched. Great muscular wasting takes place; occasionally to such an extent, that mobility is impaired or completely lost in one or all of the members. Indeed, the emaciation is extreme.

As the result of the atrophied skin being drawn tightly across the prominent bones at some points, and hanging in loose, wrinkled, flabby folds at others, the discolored or livid countenance looks prematurely old, and wears an expression of suffering, hebetude, or idiocy. The lower lip hangs, exposing the teeth most hideously, and allowing the saliva to trickle from the mouth.

Additional horror is given to the aspect of the patient when the eye becomes involved in these atrophic changes.

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[1] It is said that this anæsthesia may vary as to its *quality*, *i. e.*, may vary as the stimulus to sensation is varied, and that patients who are not able to walk with their eyes shut, or to feel a needle thrust into the skin, may appreciate the passage of an electrical current. May not this variation in quality, however, be more rationallyr eferred to a variation in degree?

A degree of anæsthesia which would preclude a patient from feeling a needle thrust into the skin, might permit of his feeling the more permeating stimulus of an electrical current.

The cornea becomes opaque, and ulcerated, the iris discolored and atrophied, and at last the whole organ shrinks into a yellowish amorphous knot.

All the mucous membranes become dry and retracted; a dry ulcer very frequently penetrates the septum nasi. From these causes the voice is altered, and thirst is usually a prominent symptom.

Such deformities as accompany nervous diseases characterized by wasting, present themselves in the extremities. Fingers and toes are distorted and stiff; the nails thin, scaly, striated, frequently lost. The withered hand, with clubbed fingers, prominent bones and tendons, is often permanently distorted into a bird-like claw, the "main-en-griffe."

After these symptoms have lasted a year or two, and usually before they have attained such intensity as that described in the last few lines, the epidermis of the atrophied skin over the extensor surface of a phalangeal joint, begins to desquamate. Then, as the result of pressure[1] exerted by the bone at this point, the skin becomes thinner, whiter, more tense and shining than before. Soon a small crack appears, or a swelling resembling somewhat a blister or boil; this breaks, sets up a process of ulceration, opens the joint, and amputates the part. Or, the bone and soft tissues on the distal side may not be amputated, but dissolved away, as it were, by necrosis with profuse discharge. The latter is said to be very common. An intermediate phalanx may be removed, as in the tubercular type.

At other times a bluish, tender, fluctuating swelling arises over some joint. This opens and pours out a sanious discharge; a deep, funnel-shaped ulcer, with discolored, thick, cartilaginous edges being formed. At the

[1] This pressure is the exciting cause in most cases; very slight injuries, in some others. In still another class the ulceration is termed idiopathic, in that no cause can be assigned for it.

bottom of this ulcer the exposed bone undergoes necrosis, exfoliates, and, after the lapse of months or years, is discharged. This form of ulceration is most common on the feet, and, as a rule, occurs symmetrically first upon one and then upon the other extremity. It is usually preceded and accompanied by chills and fever.

As I remarked when speaking of similar phenomena belonging to the tubercular form (it is still more true of this type), these processes are devoid of pain; although it is said that a certain amount is felt, occasionally, in the absorbent vessels around the part. The morbid action rarely extends beyond the wrist or ankle, and, I believe, no instance of its having reached higher than elbow or knee, is recorded.

Finally, when this stage has attained a climax, fingers and toes perish by spontaneous, dry gangrene.

The duration of the chronic form of the tubercular type is variously estimated at from eight to ten years. There is, however, a class of cases manifesting slight but indubitable symptoms, such as infiltration over the malar bones and about the nose and pendant ear lobes, that enjoys almost perfect health for a great number of years.

Toward the close the disease becomes acute and carries off the patient, or he sinks into a lethargic state, and death results from inanition, pulmonary consumption, intractable diarrhœa, or some one of the common complications.

As noticed in another connection, the anæsthetic may complicate the tubercular type at this period; then, both types persisting, the latter puts an end to life.

That life is prolonged by the supervention of the anæsthetic type seems to be the general impression.

I have had no experience with the acute form of the tubercular type. The two most rapid cases I have ever seen

occurred in the persons of two sisters, Boyens by name, patients in the Charity Hospital at New Orleans. The case of the elder terminated fatally within three years; the younger is still living, but the disease has made fearful progress. The most protracted case that I know of has lasted seventeen years, and the patient's condition, when I last saw her, was not alarming.

The average duration of the anæsthetic type is set down at about eighteen and one half years.

The patient is, as a rule, free from all complications during the major part of his illness.

As the disease advances, the victim of it sinks into a condition of depressed vitality. The skin is cold, the pulse weak, the limbs paralyzed; the nerve centres are invaded, there is great moral and physical sluggishness, and clonic, or, more frequently, tonic spasms occur. Death takes place from marasmus, tetanus, colliquative diarrhœa, or nephritis.

A few words now upon some of the more important complications.

So common are bronchitis, indigestion, and diarrhœa in leprosy, that they may rather be styled accompaniments than complications of the disease. The bronchitis of chronic and persistent type is due to congestion, or to the breaking down of tubercles in the mucous membranes of the tubes. The colliquative diarrhœa may be the product of the same causes, or result, as does the indigestion,[1] from the vitiated condition of the blood and the depression of the general health.

Albuminuria is probably, in the majority of cases, due to the invasion of the kidney by the morbid process.

Leprosy predisposes to other affections of the skin. Eczema, elephantiasis Arabum (in countries where it is endemic), herpes zona, impetigo, lichen, molluscum fibrosum,

[1] Of Van Someren's patients, 25 per cent. suffered from impaired digestive power.

pityriasis, and scabies are all on record as complications. These are worthy of note, for they may, by modifying the appearance of the disease, obscure the diagnosis. Especially is this true of scabies. Indeed, the peculiar changes produced by the presence of the acarus in a leprous skin, led several excellent observers to mistake the nature of this complication, and to declare it an eruption peculiar to leprosy, or, at least—a later opinion,—a form of itch found under no other circumstances. Owing to the state of the skin, the parasite thrives exceedingly well, and small lumps, consisting of masses of thickened epidermis, and countless numbers of the dead insects, are seen upon the hands and other portions of the body.

This disease does not protect against the exanthemata. Cases of variola and varicella have occurred in the persons of lepers in the hospitals at Bergen. Dr. Kaposi quotes from Lawrence (*Jour. Cut. Med.*, vol. i, No. 2, 1867) a case in which measles supervened during the progress of tubercular leprosy. Dr. J. H. Bemiss also gives us to understand that small-pox attacks lepers, and that vaccine matter readily " takes " on them.

Syphilis is only too often found as a co-existing plague in Norway, Sweden, the Hawaiian Islands, and other countries.

Lepers are said to be very susceptible to the influence of cold, and improper food,[1] which renders them especially liable to phthisis, erysipelas, and nephritis.

Lastly, hepatic disorders may arise in hot climates toward the end of an attack.

[1] According to Dr. Enders (*loc. cit.*) leprosy in women is usually kept in abeyance so long as menstruation is regularly performed, but the least disturbance of this function may precipitate or aggravate, should it already exist, the disease. This, however, is a *post hoc propter hoc* argument, and it appears to me more than possible that the disturbance of menstruation is rather, as in tuberculosis, an early signal of the approach of a grave constitutional disease.

A CONTRIBUTION TO THE PATHOLOGICAL ANATOMY OF LEPROSY (LEPRA ARABUM*).

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THE existence of leprosy in Louisiana in the form of an endemic disease, though not of a recent date, has of late years attracted the attention of the medical profession of this State, and thus induced me to a closer study of the pathological anatomy of this disease on three severe cases which terminated fatally in the Charity Hospital during the course of last year. The results obtained from the macroscopical and microscopical examinations of the various organs of these cases will form the chief subject of this paper; it may, however, not appear out of place to introduce it by a brief review of the history of the disease.

The general characters of this dreadful disease have been known to mankind for hundreds, nay, thousands of years, it having already existed among the ancient Jews during their captivity in Egypt, though recent authors point to Hindostan as its birthplace. From this centre it spread, in the course of centuries, over the greater part of Asia, to the South of Europe, slowly and steadily extending in a northern and western direction to Germany, France, Great Britain, Russia, and Scandinavia, and finally to America. Dur-

* Read at the Fifth Annual Meeting of the American Dermatological Association, held at Newport, R. I., Sept. 1, 1881, by James Nevins Hyde, M.D., of Chicago.

ing the Middle Ages the prevalence of leprosy among the European nations was so great, as to necessitate the establishment of numerous asylums, or lazar-houses, in every country, for the special purpose of receiving and nursing the persons affected with this disease. Since two centuries, however, the disease commenced to gradually disappear from Europe, so that at present, with the exception of Norway, leprosy has ceased to prevail upon the soil of that continent, and is there only met with in a sporadic form. But while, perhaps by the progress of civilization, it has disappeared from these countries, there are still many localities left upon the globe where it permanently dwells, or lingers in its old endemic form. Such are: Hindostan and Bengal, as well as the islands of the Indian Ocean, China and Japan, Persia, Syria, and Palestine, with the islands of Cyprus, Rhodes, Mitylene, Samos, and Crete, many localities in Australia to which it was carried by the Chinese, some parts of Africa, the island of Madeira, the shores of the Mediterranean Sea, the West India Islands, the eastern coast of South and Central America, the Sandwich Islands, to which it was also brought by the Chinese, and some other localities.

As may be presumed, a disease as repulsive and dreadful in its nature as leprosy, and to the amelioration of which the reigning authorities and philanthropists of all countries in which it prevailed appear to have contributed their share at all times, must have constantly kept awake the interest of medical men,—even in those periods of history when medical science had, as yet, not assumed the definite form in which we behold it to-day; and it is thus that the physicians of every historical period were as well acquainted with the clinical phenomena of this disease as we are at present. The investigations into its cause and nature, however, received a fresh impulse, in 1848, by the appearance of Daniellsen and Boeck's work, containing the labors of

these authors at the infected localities in Norway, to which they had been sent by the Swedish government. About ten years later, Virchow, in answering a call from the same government, visited Norway for the purpose of investigating the pathology of the disease ; and it is to him that we owe, like so many other pathological discoveries, the first accurate account of the abnormal histological changes upon which the various phenomena of leprosy depend. The labors of Virchow proved to be another stimulus to further inquiries and discussions concerning the ætiology, pathology, and treatment of leprosy, and gave rise to very numerous statistical and pathological observations, made since that time, both by appointed medical commissions, or by private physicians practising in those localities where the disease still prevails,—and a great number of excellent treatises and reports have accordingly appeared, within these last twenty years, to enrich the literature of leprosy. Every important point of this disease, therefore, has already been so thoroughly discussed as to leave scarcely any thing new to be added, unless it were to corroborate by some closer details the comparatively limited observations already made in the pathological anatomy of the organs affected by the disease.

The clinical phenomena of leprosy are so generally known from the descriptions found in text-books as to require no special notice in this place ; though it may be proper to remark that the two varieties of the disease generally described, the *tuberculated*, and the *non-tuberculated*, or *anæsthetic*, are, by many authors, no more regarded as distinct forms, but in reality depend upon the particular organs in which the pathological changes first take place. Thus, in the tuberculated form of leprosy, the neoplastic growth, represented by small proliferating cells, first appears in the cutaneous or subcutaneous tissue, giving rise to the thick-

ening, and to the formation of those characteristic knots, or tubercles of the skin, while in the anæsthetic form the cellular growth first affects the connective tissue of the nerves. The great majority of cases of leprosy belong to the tuberculated form; but, as in most of these, besides the affection of the skin, phenomena characteristic of the anæsthetic form of the disease are also observed, it has been asserted by recent authors that all cases of tuberulated leprosy would finally become anæsthetic, if the patient's life were not previously cut short by the disease, before the neoplastic elements had also made their appearance in the nervous tissues. This view concords with the cases upon which I made the *post-mortem* examinations, together with others that came under my direct observation in private practice.

The original histological element of leprosy, giving rise to the various pathological phenomena of this disease, is represented by numerous small cells, first discovered by Virchow, proliferating throughout the connective tissue of the affected organs. The exact origin of these elements seems to be, as yet, not definitely settled; for while some pathologists refer it to the cells of the cutaneous or subcutaneous connective tissue, or to that of mucous membranes, or to the interstitial tissue of other organs, others place it in the walls of the blood-vessels or lymphatics. Reserving the discussion of this point until I shall have stated the results of my own examinations, I proceed to the description of the condition of the organs of the cases above mentioned, commencing with the autopsies.

CASE I.—A girl, about 20 years of age, entered the hospital—accompanied by her sister, likewise affected with leprosy—about a year previous to her death. The mother of these women had died from the same disease; they were natives of Denmark.

Autopsy.—The skin of the face was thickened as usual, though presenting no tubercles of any remarkable size, the ears enlarged and elongated, especially the lobes; the skin of the upper and lower

extremities was in the same condition, presenting a number of tubercles, some of which were ulcerating at the time of death. The tongue was thickened, the thickening probably extending into the larynx, which, however, was in this case not examined. The conjunctiva was yellowish. When the thorax was opened the lungs collapsed to an unusual extent; their appearance was normal, though they were soft and flabby. The heart small but normal. In the abdomen, the tributaries of the portal vein were found congested. The stomach and intestines were highly congested throughout. The duodenum and other portions of the small intestines presented an intense yellowish brown tint, that of bile, which, together with the red tint of the congested blood-vessels, rendered the aspect very peculiar. The greater number of the glandular patches of Peyer, as also many of the solitary glands, were found diseased; they were swollen and of a brownish tint, the affection increasing the nearer they were placed to the termination of the ileum. Some small ulcers were met with in the ascending and transverse colon. All the mesenteric lymphatic glands were greatly swollen and rendered blue by the congestion. Those placed alongside the vertebral column—both in the abdomen and thorax—were in a similar condition. The liver was normal in size, but soft in consistence and yellowish in tint. The kidneys were smooth, normal in size and form, but flabby in consistence; when cut, their cortical substance presented a pale, though not yellowish tint. The spleen was almost smaller than normal, but of a narrow and elongated form, the color of its surface and parenchyma normal. The suprarenal bodies normal in color, but narrow and elongated in form; the bladder normal. The walls of the aorta—especially in the abdomen—and of the iliac arteries greatly thickened, and the calibre of the vessels diminished; the latter condition was observed on the arterial trunks arising from these vessels. The surface of the brain, with the exception of some vessels of the pia mater being filled with blood, presented a normal appearance; but when cut, the surface of the section appeared rather anæmic. The spinal marrow was not examined. The semilunar and other ganglia of the solar plexus were found very small and soft, atrophied.

CASE 2.—A middle-aged man, native of Louisiana, French descent.

Autopsy.—Lungs normal. Heart normal in size; tricuspid and mitral valves thickened throughout by neoplastic matter in the

form of nodules ; corpora Arantii of the semilunar valves of the aorta considerably enlarged, but the valves themselves, as also those of the pulmonary artery, of a healthy appearance. A considerable portion of the intima of the latter and of the arch of the aorta presented a scarlet color (endo-arteritis). The wall of the left ventricle, when cut, showed a darker color than normal. The alimentary canal, as in the first case, was found congested throughout, the solitary and conglomerated glands greatly swollen, many of them exhibiting a brownish tint. The lymphatic glands of the abdomen—the mesenteric included—were also greatly enlarged, though not blue from congestion. The liver was normal in size and almost of a normal color, though rather soft and in many places exhibiting a pale yellowish tint ; the gall-bladder contained a golden-colored bile. Of the kidneys the one was of natural size, but misshaped ; when longitudinally divided about two thirds of the cortical substance was found intensely congested, while the remaining third exhibited a yellowish tint. The other kidney was below the normal size, and still more misshaped by consisting of three larger and two smaller lobes ; its vessels were issuing from the furrow formed at the place of junction of the lobes upon the flat side of the organ ; when divided it presented a yellowish tint with a narrow red border directly under the capsule. The spleen was greatly enlarged, nearly twice the normal size, and misshaped, though normal in color and consistence ; when cut the parenchyma exhibited a normal appearance. With the exception of many of the larger vessels of the pia mater being filled with blood, the brain presented nothing abnormal, nor did the larger nerve-trunks ; the spinal marrow was not examined.

Case 3.—A man, between 30 and 40 years of age, native of Louisiana, French descent. In this case the skin was affected to a great extent ; there were a number of ulcers present upon the forearms, hands, legs, and feet, from which, during life, a horrible odor arose. Upon the mucous membrane of the nose the disease had very considerably advanced, destroying portions of the nasal cartilages, and causing the nose to bend inward. Six months before the patient's death, tracheotomy had been performed to relieve him from suffocation.

Autopsy.—The mucous membrane of the epiglottis and larynx was greatly thickened and ulcerated. The posterior and inferior portions of the lungs were congested, the rest normal. The

heart was normal, nor did the large blood-vessels appear to be affected. The mucous membrane of the alimentary canal was only slightly congested, though the mesenteric lympathic glands were, as in the preceding cases, much swollen; the same condition prevailed upon the other abdominal and the thoracic lymphatic glands, while those of the femoral and inguinal region had attained an enormous size, presenting a dark bluish and brown color. The liver was rather enlarged and presented a mottled appearance of dark blue and yellowish spots, indicating both congestion and fatty degeneration; its surface was rather rough. The kidneys were normal in size and form; some portions appeared congested, while others presented the yellowish tint. The spleen was normal. The vessels of the pia mater, as also those of the brain substance were congested, but no other lesions upon this organ were observed at the autopsy. The vessels of the spinal marrow were considerably congested; the cord itself, especially in the cervical region, appeared swollen. The Gasserian ganglion was found considerably indurated, and smaller than normal. No thickening of the connective-tissue sheaths of the larger nerve-trunks could be discovered.

The above statement shows that these three cases resembled each other in the macroscopical pathological changes observed upon the various organs, the condition of the latter fully corresponding to that of other numerous cases described by various authors. In proceeding, now, to the microscopical examination, I shall commence with that organ upon which the particular changes characterizing the disease are first observed, namely, the skin.

It has already been remarked that the characteristic histological element of leprosy is represented by certain small proliferating cells of a round, oval, uni- or bipolar, or otherwise irregular form. The origin of these cells is generally referred to the cells properly belonging to the connective tissues of the affected organs, such as the skin or mucous membrane, or of the adventitia of the vessels, the sheaths of nerves, the capsules, etc., but, as I shall show directly, the neoplastic cells may also be derived from the glandular, epi-

thelial, endothelial, and even fat-cells. While many of them may be observed single, the greater part of them, especially in tissues where the pathological process has been in operation for some time, are met with in the form of groups, consisting of from two to a dozen, or even more, of individuals, each group representing the progeny of the original cell. The form of these groups, though almost always irregular, is generally elongated, oval, pyramidal, or ellipsoidal; even those groups derived from the nuclei of the fat-cells finally assume an irregular, oblong, or ellipsoidal form.

For the study of these cells in the skin, the thickened ear is probably one of the most suitable parts for making thin sections, especially the lobulus, as it represents two layers of skin united by the subcutaneous tissue, the panniculus adiposus.

In examining, then, a thin section taken from the lobulus of the ear—which, in old lepers, is always found very considerably enlarged,—the characteristic cellular element may be studied in its various stages of development. Judging from the results of the examinations which I made of different portions of the ear, or other parts of the skin, in the three cases under discussion, it appears that the disease commences in the pars reticularis of the corium, whence it may proceed, in one direction, and invade the pars papillaris, and even extend throughout the mucous layer of the epidermis, or, in another direction, to invade the subcutaneous tissue; but it may also extend in both directions at the same time. In the sections which I examined, especially those of the lobes of the ear, the disease had always invaded the subcutaneous tissue, in one case even all the fat-cells, while in many places of the section the pars papillaris, and in consequence also the epidermis, had remained free, and showed a perfectly normal structure. In other places, on the other hand, not only the papillary layer of

the corium, but also the stratum mucosum of the epidermis had been transformed into the neoplastic tissue. The individual cells representing the latter show an average diameter of about .008 to .01 mm., and contain a round nucleus of about .005 mm. in diameter, with a distinct double contour, in the interior of which a number of small granules are observed. In some instances a narrow constriction, or dividing line, is observed upon the nucleus, or the latter is already divided, so that two of these bodies are found enclosed within the cell. After the division of the nucleus, that of the protoplasm of the cell takes place. But though the boundaries of the cells, thus arisen, are in most instances distinctly seen, the cells themselves are not always observed detached from each other; on the contrary, the process of division may take place again on these new cells, and continue during their growth until a large group, as already mentioned, is formed. A close examination of such a group will show that, though bounded by an unbroken outline, it represents not a single cell containing a number of nuclei, but that each of the latter is surrounded by protoplasm, representing an individual cell. The nucleus, however, is not always distinctly seen in every cell of the group, but frequently hidden by the protoplasm.

It has been stated by some authors that these neoplastic cells take their origin from the cells or nuclei of the blood- or lymphatic vessels; this, however, appears not to be the case, for, though the number of nuclei contained in the walls of these vessels, especially those of the adventitia, really appears in some instances to be increased, the increase forms rather the exception to the rule; nor do the cells proliferate to a greater extent in the close vicinity of these vessels than elsewhere.

The external or fibrous layer of the hair-sacs, also, appears to be rarely invaded by the proliferating cellular ele-

ments; in most instances I have observed it to present a perfectly normal appearance, even when closely surrounded by the proliferating cells. Different it is with the external root-sheath, which is frequently observed to have gained in thickness, and to be completely separated from the internal root-sheath, which, in its turn, is detached from the cuticle of the hair; in some places, also, the last named root-sheath appears thickened. In some instances the hair itself has remained attached to the papilla, while in others it is detached and found some distance from the latter. Many hair-sacs present—always below the neck—a considerable dilatation, or varicosity, resembling a commencing invagination confined to the external root-sheath and the fibrous layer of the sac. By this dilatation the empty space between the external and internal root-sheaths is considerably increased, though the two layers involved are usually thickened in the vicinity of the dilatation mentioned. It is possible that this phenomenon is produced by a contraction of the pars reticularis of the neighboring corium.

Though traces of the ducts of the sudoriferous glands are not often met with in the sections, the coils of the glands themselves appear to generally remain unaffected, their secreting cells mostly presenting a normal appearance; in some instances only the cells of the connective tissue surrounding the coil appeared enlarged. On the other hand, the secreting cells of the sebaceous glands are frequently involved in the general infection, and the process of cell-proliferation commences, as in the corium, by a division of the original nuclei, continuing until each individual gland-cell has been converted into a number of the characteristic smaller cellular elements. In sections stained with picro-carmine the particular pathological condition in which the latter are met with is easily distinguished by the color, for while, in some places, they appear stained with carmine,

they exhibit the yellow color of the picric acid in others, indicating the retrogressive metamorphosis which they are undergoing. Sometimes one or more nuclei, or even entire cells, of a group exhibit the ordinary uncolored refractive appearance of fat in the form of a smaller or larger globule; but frequently the whole group of cells may also be found converted into a large single fat-globule. This singular behavior of the protoplasm of these cells I am unable to explain satisfactorily, though it is evident that the absorption or non-absorption of the picric acid by the former is indicative of different stages in the degenerative process of the cells. The phenomenon is, however, not only observed on the secreting cells of the sebaceous glands, but also on those of the corium, and on others to be mentioned hereafter. The acini of these glands are frequently met with filled and dilated with fat.

In the fat-cells the pathological process commences, as in the other tissues, by a successive division of the nucleus, giving rise to the formation of new cells, which, in most instances, remain attached to each other in the form of a smaller or larger group. They appear to proliferate along the membrane of the original fat-cell, absorbing it during their growth. It is thus that, frequently, individual fat-cells are met with, a large portion of the surface of which is represented by the proliferating cells, whilst the rest has remained unaltered, and presents the ordinary refractive appearance by virtue of the portion of normal membrane left, as well as of the fat still inclosed. In some of these instances the cells are stained with carmine, showing that the fatty metamorphosis has, as yet, not commenced, though generally they have already absorbed the picric acid, indicating that the degenerative process is going on. The small groups of fat-cells lodged in the areolæ of the pars reticularis of the corium, almost always undergo the

characteristic change just described; they had done so, at least, in all the sections of skin which I examined. The same happens with the larger groups in the subcutaneous tissue, if the patient lives long enough. In the sections extending throughout the lobulus of the ear of Case I not a single normal fat-cell could be discovered, whilst in the other cases a few small groups had been left unaffected in the subcutaneous tissue. In the neoplastic groups of cells, originating from the nuclei of the fat-cells and destroying the latter, the phenomenon of fat-globules making their appearance is observed in the same manner as in those of the corium or of the sebaceous glands.

In sections made through the helix and anti-helix of the ear the same conditions and changes above described were likewise observed to prevail, though they extended not to the perichondrium and cartilage, both of which presented a normal appearance. The same remarks are applicable to the mucous membrane and subcutaneous tissue of the thickened epiglottis. Here the neoplastic growth presented exactly the same characters as in the skin, invading all the parts, including the epithelial cells of the racemose glands, but leaving the perichondrium and cartilage, as well as the fat-cells, untouched. Upon the anterior surface of the epiglottis the mucous membrane was found almost entirely detached from its subcutaneous tissue, very probably by the softening of the tissues.

In the tongue the pathological changes had not proceeded to so great an extent as in those parts already mentioned. The proliferation was chiefly confined to the perimysium of its composing muscular bundles, and to the connective tissue surrounding the vessels, though the mucous membrane was also found slightly involved in these changes. A few of the blood-vessels showed an increase of the nuclei of their adventitiæ.

As already mentioned in the first of these cases, the aorta and other large arteries were found very considerably affected by the disease, resulting not only in a thickening of their walls, but, moreover, in a general reduction of their normal calibre. The microscopical examination of thin horizontal sections of the aorta showed that, as in the other tissues, the thickening depended upon a proliferation of the nuclei, particularly of those belonging to the muscular fibre-cells. In these, however, the neoplastic growth appeared to be mainly represented by proliferating nuclei, generally appearing in small oblong or ellipsoidal groups of two or three individuals, their axes lying at right angles with that of the muscular fibres; larger groups of these nuclei were, however, also here and there observed. In some places, in the media of the vessel, the proliferation had been going to such an extent as to almost fill up the interior of these fibres by long rows or masses of nuclei, appearing highly stained with carmine, while the protoplasm of the fibre-cells presented the ordinary staining, and the elastic-tissue fibres had remained perfectly colorless. In the adventitia of the vessel the proliferation of the nuclei had only proceeded to a very small extent; it therefore presented a normal appearance in general, while in the intima the pathological changes had in many places given rise to the fatty metamorphosis, and to a complete disorganization of the membrane by softening and ulceration. The proliferating elements here appeared mostly in the form of round cells, having undergone fatty degeneration and resembling pus-cells; though in some places numerous nuclei and cells more or less stained with carmine were observed in the different layers of this membrane. As in the media, the extent of the morbid changes differed in different places of the intima, the degree of thickening of the wall of the vessel being proportionate to that of the

proliferation. On the whole, the pathological process here greatly resembles that of chronic endo-arteritis.

Before proceeding to the description of the pathological changes observed in some of the glands, and in the nervous tissues, some brief remarks regarding the staining and examination of the sections of the organs may here not be out of place. In the study of the neoplastic growth of leprosy, as in other histological investigations, much advantage is gained by staining the sections with two or three colors for the purpose of defining different conditions of the anatomical elements of the tissues. The picro-carmine of Ranvier and the alum-hæmatoxylin I have found the most suitable to meet the object in view, for while the carmine is only absorbed by the normal protoplasm of the nuclei and cells, the picric acid, moreover, stains the fatty substances, and the hæmatoxylin imparts a deep blue to the nuclei before coloring the protoplasm of the cells. Thus, in staining the sections with picro-carmine only, the commencing degeneration or fatty condition of the protoplasm is easily recognized by the yellow staining of the picric acid, while the normal protoplasm will appear carmine, and the fully formed fat not appear stained at all, but manifest itself by its ordinary refractive appearance. The nuclei of the cells, though recognizable, do not always appear very distinct; but, if the section is subsequently put in a weak solution of hæmatoxylin, and left there only sufficiently long to stain the nuclei alone, these bodies will be very easily distinguished by their blue color from the other elements; though, if the section is left too long a time in the staining liquid, the protoplasm of the cells will be rendered dark purple, or even blue, by the hæmatoxylin hiding or driving out the carmine. Another advantage of the subsequent staining with hæmatoxylin is, as I have found, to enable one to distinguish the comparative age or

freshness of the protoplasm by the degree of intensity of the staining; in other words, the more recent the histological product the more readily and intensely it will be stained. The proliferating nuclei, therefore, will be found more highly stained than those of the normal tissues.

In the same way fibrinous products, such as thrombi, or emboli, in the blood-vessels, will be found highly stained by hæmatoxylin, and rendered more distinct for recognition. The examinations should not be confined to sections mounted in one and the same medium, such as Canada balsam. This substance renders the preparations remarkably clear and transparent, and, provided they are perfectly stained, offers many advantages in their examinations, consisting, especially, in obtaining a correct idea of the relationship of the anatomical elements of a tissue to each other, of which, in virtue of the transparency, a more perspective view is obtained. For the study of the more minute details of a tissue, however, the Canada balsam is unsuitable, as, in proportion to the transparency gained, definition is lost. While, therefore, some sections may be mounted and examined in Canada balsam, others should, at the same time, be examined in glycerine, which renders the tissues sufficiently transparent to show their minutest details. In the examinations themselves great advantage will be derived from using oblique illumination by the achromatic prism, for the application of which, however, only very superior objectives with high angular apertures are suitable.

In continuation of the description of the microscopical condition of the organs examined in the cases of leprosy under discussion, I shall now proceed to the liver. The pathological changes observed in this organ presented the same general characters in all three cases, the difference observed consisting only in the degree of their extent. The pathological process, as in the tissues already described,

here also consisted in the characteristic proliferation of the cellular elements, affecting not only the connective tissues, but, moreover, the hepatic cells themselves, and even the endothelium of the intra-lobular hepatic veins. In the first case the proliferation of the connective-tissue cells was chiefly confined to the walls of the intra-lobular veins, from which the morbid process had extended to the endothelial cells of these vessels, manifesting itself by the division of their nuclei and their ultimate fatty degeneration. In consequence of these changes, the walls of the vessels were slightly thickened and their lumen decreased in size. The portal vessels and their capsule, on the other hand, appeared in this case not much affected,—only in the latter a slight proliferation of cells was observed.

The greater portion of the secreting elements—the hepatic cells—had received their full share of the infection, and were met with in the various stages of fatty degeneration. The degenerative process appeared to have mostly extended through that portion of the lobules farthest removed from the vessels, only in some places the degeneration had extended to the cells in the vicinity of the interlobular vessels, while those forming the central portion of the lobule and surrounding the intra-lobular (hepatic) veins, were mostly found perfectly colored by the carmine. Throughout the sections a number of cells of a brown color, and containing a number of highly refractive, dark-bordered granules, were observed. The brown color of these cells was probably due to the presence of bile, and as the granules had remained unaltered, even in cells where the protoplasm had undergone fatty degeneration, they very likely represented pigment. In the degenerated portions of the lobules, a number of the cells were found colored yellow by the picric acid, while others were colorless, or contained one or two large ordinary fat-globules

great numbers of the latter, however, were also found in connection with the other cells, wherever the degenerative process was going on, and even amidst the cells of normal appearance, that is, those stained with carmine, many of which contained one, two, or more fat-globules, probably derived from the fatty degeneration of the nuclei. On these cells, also, the beginning of the process could be observed manifesting itself, as usual, by the division of their protoplasm and the presence of two or more nuclei, though in those cells already undergoing the fatty metamorphosis the nuclei could be no more distinguished. In this liver, as may be judged from the above sketch, the degenerative process had already advanced to a considerable extent, moreover indicated by a certain faintness and delicacy observed in the outlines of all the minute anatomical elements of the organ.

In the second case, though the same changes, as above described, were observed in connection with the hepatic cells, the disease manifested itself more strikingly upon the connective tissues of the organ. Thus, the capsule of the portal vessels (Glisson) was found affected and considerably thickened throughout, and encroaching upon the neighboring hepatic cells. The thickening, however, did not evenly extend throughout the whole capsule, but was much greater in certain places, where, in thin sections, it appeared in the form of bulgings projecting into the neighboring parenchyma. While the adventitia of the inter-lobular vessels was likewise undergoing these changes, their other coats, as also those of the ducts, appeared unaffected. But the proliferating cells of the adventitia and capsule, instead of being transformed into a so-called granulation or cicatricial tissue, as takes place in the ordinary cirrhosis of the liver, here rather manifests a tendency to an early fatty degeneration; and, accordingly, as in the corium of the skin,

these elements were also met with in various forms and conditions, either singly or in groups, unchanged, or containing large and distinct fat-globules. It is to this circumstance, that the softness of the liver in leprosy, and the want of the knotty appearance produced by the organization and contraction of the granulation-cells, as observed in the so-called "hobnail-liver," must be attributed. In the capsule and walls of the intra-lobular veins the morbid process had proceeded to a still greater extent, involving the endothelial cells, the proliferation of which finally led to an obliteration (thrombosis) of the lumen of these vessels. There was hardly an open intralobular vein met with in the sections examined of this liver. It remains to be stated that in the instances just described the remains of the connective tissue of the capsule or adventitia could always be seen defined to a certain extent around the lumen of the vessels. There were certain masses of cells met with in these sections, however, which perfectly corresponded in their details with those presented by the horizontal or transverse section of a small intralobular vein, but of which the outlines bordered directly on the surrounding hepatic cells without showing any definite traces of connective-tissue fibres, though the mass appeared to be crossed by apparently fibrous elements. I am unable to decide satisfactorily upon the true nature of these bodies, unless they represent small centres of proliferating hepatic cells, the appearance of fibres being caused by the empty capillaries stretched throughout the mass. The epithelium of the hepatic ducts, as seen in horizontal sections, generally appeared highly colored by the carmine, indicating its normal condition. In this case, also, numerous brownish hepatic cells with dark-bordered granules were met with throughout the sections. The outer capsule of the organ was always found in a normal condition.

In the third case the same pathological changes observed in the liver of the second were also met with. In addition to these, however, the minute vessels of the organ were found congested; in many places, even, to such a degree as to become completely filled with blood-corpuscles and free hæmoglobin, the presence of the latter indicating a stasis. This condition fully explains the dark bluish appearance which the organ presented at the macroscopical examination.

From the presence of the neoplastic element in the connective tissue of the liver, it might be inferred to be likewise met with in the kidneys. This, however, as will be seen directly, is not the case; for these organs, at least in the cases under discussion, appeared to form an exception to the rule. The chief pathological phenomenon observed here consisted in the formation of albuminoid cylinders in the uriniferous tubules.

Thus, in the first case, a considerable number of these cylinders were observed in all the different portions of the tubules; many of them consisted entirely of the albuminoid substance, while in others epithelial cells were seen to be embedded. These cylinders possess the power of absorbing carmine, or other staining material, in a high degree, and are in consequence easily distinguished by their staining and lustrous appearance. The epithelial cells of the tubules, in this case, had generally preserved their normal size and appearance, with the exception of a small number in which they presented the same high carmine coloring, or staining, as the cylinders. On a former occasion, in connection with a discussion on the pathological changes observed in the yellow fever kidney, in which the same phenomenon is observed, I have dwelt at length upon this subject, expressing the view that these particular cells represented the initial stage of the retrogressive metamorphosis,

containing the abnormal material of which the cylinders were subsequently formed, a view which has been advanced of late years, and is now held by a number of prominent pathologists. The epithelial cells covering the glomeruli also presented the intense staining and lustrous appearance before mentioned. In some places of the sections a number of cells were likewise observed, the protoplasm of which had remained uncolored, while their nuclei had absorbed the carmine, though no fat-globules could be detected. In the interior of some capsules from which the glomeruli had fallen out, a pale carmine-colored exudate was moreover observed. The larger and smaller blood-vessels throughout the sections were generally found empty of blood-corpuscles, though in certain places of the medullary portion of the organ small portions of the capillary network, as well as some arterioles, were met with containing small fibrinous emboli, intensely colored blue by the hæmatoxylin; in these arterioles the endothelial cells were, in some places, also colored by this agent. As already mentioned, there were no traces of the characteristic neoplastic element observed in the connective tissue of this organ, neither in the walls of the blood-vessels, nor in the interstitial tissue; they everywhere presented their normal appearance.

In the second case, with the exception of the emboli in the blood-vessels, the same changes as just described were observed; the exudate from the epithelial cells covering the glomeruli was rather more abundant.

As in the liver of the third case, in its kidneys, also, the pathological condition of these organs presented itself in a still severer form than in the preceding cases. For, though here the albuminoid cylinders were not as numerous, the epithelial cells of the uriniferous tubules had in many places undergone a complete fatty degeneration, the degenerated individual cells being represented by fat-globules,

which, in many instances, presented the original form of the entire cell. Besides, the lumen of many tubules was filled with fat. The cells of the epithelium covering the glomerular vessels presented a high staining, as in the other cases. The blood-vessels, especially the straight arteries of the medullary substance, were found filled with blood-corpuscles and free hæmoglobin, and extensive extravasations of this coloring substance had taken place from the vessels into many of the tubules, to the cells of which it had imparted an intensely brown color. With all these changes, no proliferating cells could be detected in the walls of the blood-vessels, or in the interstitial tissue.

The pathological changes observed in the lymphatic glands greatly resemble in their nature, those already described as taking place in the liver. In the third case, they were observed to have advanced farthest, for which reason I shall place this case first in the following description.

While the outer stratum of the connective tissue of the capsule investing the glandular tissue, namely, showed a normal condition, the inner layer, with the trabeculæ arising from it, presented great numbers of the proliferating neoplastic cells in smaller or larger groups and distinguished by the same characters as those met with in the skin or in the capsules of the inter-lobular and intra-lobular veins of the liver. Many of these elements had undergone fatty degeneration, indicated by numerous, generally large fat-globules. In the follicular substance of these glands, also, the same proliferating cells were met with in great numbers; though here the successive stages of the retrogressive metamorphosis, as in the sebaceous glands of the hair-sacs and racemose glands of the epiglottis, could be distinguished by the difference in the staining of the elements concerned. Accordingly, some of the groups of cells were found stained with carmine, while others presented the yellow color of the

picric acid, indicating that the retrogressive changes had commenced, and others, finally, contained large refractive fat-globules. The follicular columns themselves appeared enlarged by the proliferating neoplastic elements. Whence the latter were derived, that is, from the lymph corpuscles of the follicular tissue, or from the cells of its reticulum, remains difficult to decide; but, judging from the great number of those cells, it may be presumed that the lymph-corpuscles had been involved in the pathological process. In the reticulum of the lymph-tracts the proliferating element presented a new feature, for here it was represented by yellow or brown, highly refractive, mostly round, oval, or oblong bodies, consisting of small round nuclei, distinguished by a double contour, and by containing a few small granules in their interior. In many places, especially in the vicinity of the capsule and the hilus, these bodies filled up the entire lymph-tract, and, by virtue of the mass, they then presented a deep brown color. The larger ones appeared unattached to the reticulum, and, in consequence, not derived from the nuclei and cells of this structure; a number of the smaller, however, were directly observed to represent the nuclei of the multipolar cells, forming the reticulum of the lymph-tracts; they were of a light yellow color, and appeared highly refractive. As in the follicular tissue, here also some of these bodies, especially those unattached to the reticulum, may have originated from the nuclei of free lymph-cells. As in the outer stratum of the capsule, the cells of the connective tissue, surrounding the blood-vessels in the hilus, had not been affected by the proliferation-process, though the latter had extended to the walls of the lymphatic vessels; the blood-vessels, also, presented a normal appearance. Judging from the condition above described, the lymph-current in these lymphatic glands must have been entirely arrested.

In the lymphatic glands of the first and second case the same pathological phenomena were observed, the difference met with existing only in the smaller extent of the morbid process.

In the medullary substance of the supra-renal bodies a considerable number of cells were also observed to have multiplied into groups and undergone the same retrogressive changes as observed in the tissues already described.

In the spinal marrow of the third case the proliferation of cells was only met with in the ependyma of the central canal, which, in some places, was even found to be occluded. A number of the nuclei of the posterior commissure in the vicinity of the canal, also, were observed to have multiplied. These changes were found to extend along the entire central canal up to its orifice in the fourth ventricle. The nuclei of the neuroglia, on the other hand, had remained unaffected. But while the connective element had thus far preserved its normal condition, considerable portions of the nerve fibres, particularly in the posterior, but also in the other white columns, had undergone degeneration. The axis-cylinders of these fibres had absorbed no color, and the medulla of many of them appeared swollen and increased in diameter. This condition existed throughout the whole cervical region, and was even met with, though in a much lesser degree, in the lumbar region.

In the medulla oblongata, also, many bundles of nerve fibres manifested this state of retrogressive metamorphosis by the absence of their absorbing power for the staining material, while a number of ganglion-cells were observed to have undergone a pigmentary degeneration, the whole ganglionic body representing a mass of brown pigment granules. The smaller blood-vessels, both in the medulla oblongata and the spinal marrow, were found filled with blood corpuscles.

In some parts of the corpus striatum, a considerable proliferation of nuclei was observed in the ependyma, causing a number of ridge-like elevations, or folds, upon this layer. The same proliferation was observed of the nuclei of the neuroglia throughout the whole ganglion, where they appeared in groups of two, four, or more. While the nuclei of the ganglion-cells appeared perfectly stained and normal, the protoplasm of these bodies had refused the staining material, and appeared colorless.

No particular changes were observed in the sections made of the cortex cerebri of the central convolutions.

The Gasserian ganglion was, as already mentioned, indurated and smaller than normal, its investing connective-tissue sheath thickened and adhering to the dura mater. The induration and thickening was due to a proliferation of the nuclei of the connective tissue, extending, however, into the ganglion itself. The same phenomenon was observed on the semilunar and other ganglia of the solar plexus.

The changes observed in the nervous tissues of this case, though sufficiently severe, were not quite as extensive, as they have been met with by other observers in some other cases; thus, the connective-tissue sheath of the larger nerve-trunks, in which, in a number of instances, the neoplastic element was met with by other observers, had here remained unaffected. The lesions in the spinal marrow, also, have in several cases been observed in a more severe form. In a case of lepra anæsthetica, Steudener found in the spinal marrow a narrow cavity, filled with a viscid mucoid fluid, and formed at the expense of the gray substance. Langhaus found in the spinal marrow softening and atrophy of the commissure, the columns of Clarke, and the posterior horns, especially in the cervical enlargement and in the upper dorsal region. In the peripheral nerves he found

thickening of the peri- and endoneurium, with atrophy of the medullary sheaths, though the axis-cylinders had been preserved. Tschiriew observed in the central canal of the cervical portion of the spinal marrow many lymphoid embryonal cells of a round form, infiltrating, also, the walls of the canal. The same elements he met with in the gelatinous substance of the posterior horns. In the left posterior horn he noticed small hemorrhages and extravasated colored blood corpuscles, and also a diminution and alteration of the ganglion-cells.

The larger veins, also, on which, in the above-described three cases, no particular change was observed, have been found affected in a few other cases. Thus, in Steudener's case, the brachial vein, at the middle of the upper arm, was found considerably thickened and obstructed by a puriform disintegrated thrombus. Bœttcher observed on the veins of the forearm a thickening of the adventitia, caused by the proliferation of the small cellular elements. The muscular coat, also, had increased in thickness, causing a diminution in the lumen of the vessels. Moxon, also, described a case of leprosy in which small knots, filled with a pus-like liquid, were, in numerous places, found connected with the walls of the veins of the forearm and hands.

The neoplastic cellular element, characteristic of leprosy, has also been observed by Bull and Hansen to occur in the cornea and iris; in the former the growth generally affects the periphery, leaving free the centre.

Hansen, who has largely contributed to the pathology of leprosy, described, as early as 1870, certain round, oval, or spindle-shaped cells, containing besides the nucleus one or more larger or smaller round granular masses of a yellow color, which generally absorb carmine. These elements he met with, not only in the leprous tubercles of the skin, but also in the affected lympathic glands, the interlobular tis-

sue of the liver, the spleen, and even in the choroid and retina of the eye. To these elements, which he appears to regard as characteristic of leprosy, he refers again in his more recent contributions. In all the sections of the organs which I have examined, I have not been able to detect any cells resembling those described by Hansen, unless they are identical with the refractive, yellow nucleated masses, which, as already stated, I observed in the lymph-tracts of the lympathic glands.

Amyloid degeneration has been stated to occur in the liver and kidney, though no traces of this process were met with in the cases which I examined. Boettcher observed, in his case, upon the cut surface of the liver, numerous white dots upon the ramifying branches of the portal vein ; those are, very probably, identical with the bulgings I have described above, formed by the neoplastic cells in the capsule of the interlobular veins.

From the description rendered above it may be gathered that the results obtained from my examinations correspond, in the main, with the statements of other observers. It will, however, be noticed that, while the latter have generally described the neoplastic element as limited to the connective tissue, my observations show that the proliferation also takes place on the cells of glands and of the endothelium of vessels, such as I observed in the sebaceous and lymphatic glands, the liver, suprarenal body, and the endothelium of the intralobular veins. The whole pathological process, therefore, can hardly be regarded as a mere hyperplasia of the connective tissues, but its products, as it appears to me, represent rather a neoplastic growth, resembling in its general character the tubercle of tuberculosis, ultimately undergoing, like the latter, a retrogressive metamorphosis. The neoplastic cellular element of leprosy, remaining for a long time limited to the skin, and being probably also of a

slower growth than the tubercle of tuberculosis, does not immediately endanger the life of the patient; and it is for this reason that the disease may extend over a period of twenty or even thirty years, before it leads to death. The fatal termination of leprosy depends, undoubtedly, mostly upon the neoplastic cells appearing in the internal organs, especially the lymphatic glands and liver; the affection of the former depraving the blood, and, in consequence, interfering seriously with the nutrition of the organism, while the organic disease of the latter gives rise to the pathological phenomena observed in the alimentary canal, almost always present in the later stages of the disease, and taking a share in the general exhaustion of the patient. To the abnormal nutrition of the organism, also, must be attributed the retrogressive changes observed in the nerve fibres of the spinal marrow and brain, as they can hardly be caused solely by the proliferating cells in the walls of the central canal. The congestion of the vesssels of the cerebro-spinal axis is probably due to a neuro-paralysis of its blood-vessels, while the anæsthesia of the skin and muscles may partially depend upon the presence of the proliferating cells in the connective tissue of the peripheral nerves.

THE STRUCTURE OF THE UPPER END OF THE FEMUR, AND SOME SPECIMENS OF ACCIDENTAL DEVIATIONS OF THIS STRUCTURE.

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[During the delivery of this lecture the original specimens of normal and deviated structure of bone were used for illustration. In this report for these ARCHIVES photo-lithographs of these specimens have been employed, thus changing the phraseology, but not the meaning of the text.]

GENTLEMEN: I am here this morning to talk over some points in regard to fracture of the neck of the femur. The word *over* expresses what I mean; for we have been through the material before us, some of it at any rate. And now I propose to go through the matter again, and present you with some new material.

In the first place, let me direct your attention to the neck of the femur and its capsule. It is sufficiently accurate to say that the neck of the femur is bounded above by the femoral head. The base of the neck of the femur is bounded, in front, by the spiral ridge; posteriorly, by the posterior intertrochanteric ridge; that irregular space which I show you between these two ridges above, constitutes the superior boundary; and, roughly, a line drawn between the lower ends of the intertrochanteric ridges marks the inferior boundary of the base of the femoral neck.

Please to observe that I use the word *ridge* instead of line to designate the elevation of bone at the base of the

femoral neck in front. It is more appropriate. So much for the femoral neck; and we must not violate our definition as we go on in our work.

The upper end of the cervical capsule of the femur is attached to the circular eminence of the acetabulum. The lower end of the cervical capsule of the femur is attached (1) in front, to the base of the spiral ridge; (2) posteriorly, to the middle of the femoral neck; (3) above, by a line running from the middle of the femoral neck to the upper end of the spiral ridge; (4) below, by a line running from the lower end of the spiral ridge to the middle of the femoral neck. Sometimes more and sometimes less than one half of the femoral neck is covered posteriorly by the cervical capsule. Almost always the entire femoral neck is covered in front by the cervical capsule. So much for the cervical capsule, and we must not violate our definition. But we shall soon see the bearing this definition will have on the subject of fracture of the neck of the femur.

The logical sequence coming out of these facts is: (1) In a rare instance, where there is some space between the spiral ridge and the basal insertion of the cervical capsule, there could be an *extracapsular* fracture of the neck of the femur. In any other instance an extracapsular fracture of the neck of the femur would be impossible. (2) In another instance, when the outer and lower half of the femoral neck is broken, the fracture will be *intra*capsular *in front* and *extra*capsular *behind*. The same conclusion will hold good when the femoral neck is driven into the cancellous tissue of the trochanters. (3) In another instance, when the inner and upper end of the femoral neck is broken, the fracture will be *intracapsular*.[1]

[1] Since writing the above paragraphs I have found three specimens in which the capsule was adherent to the anterior surface of the femoral neck for at least three fourths of the distance from the anterior trochanteric ridge. In one of these there was a fracture of the outer half of the femoral neck, and therefore a veritable *extracapsular fracture of the neck of the femur*. But how can we know that we have such a case without a *post-mortem* examination?

In order to have a better idea of the subject under consideration, it is important to keep in mind some points in regard to the intimate structure of the upper end of the femur. To show this structure the upper end of the femur is cut into sections in three different directions, namely:

1. In a longitudinal direction, so that the sections will be vertical (see fig. i). The figure is drawn from a bisection of a normal femur, and shows the manner in which the compact tissue runs up to the head of the bone on the inner and under side of the femoral neck, becoming thinner as it sends off plates of bone and approaches the base of the head. On the outer side of the femur the compact tissue also becomes thinner as it sends off plates of bone and forms the great trochanter. The wedge-formed compact tissue on the inner and under side of the femoral neck, marked 1, may be called the *femoral brace*.

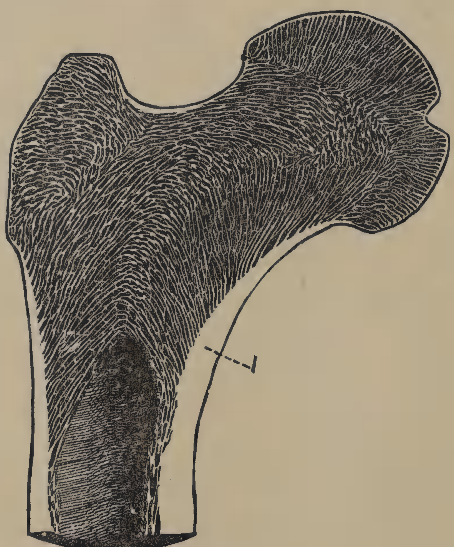

FIG. I.

2. The sections are also made in an antero-posterior and longitudinal direction. See figs. ii, iii, and iv. (a.) Fig.

ii represents the upper end of the femur, having about one fourth of the head and neck and great trochanter removed from the upper side. The part marked 1 is above the lesser trochanter, and the part marked 2 shows the cancellous tissue of the spiral ridge. The compact tissue of the neck is thin. (b.) Fig. iii represents the upper end of the femur, having about one half of the head and neck and great trochanter removed from the upper side. The part marked 1 is the upper end of the lesser trochanter, and the part marked 2 shows the cancellous tissue of the spiral ridge. The part marked 3 shows but very little compact tissue. The compact tissue of the neck begins to be thicker. (c.) Fig. iv represents the upper end of the femur, having about three fourths of the head and neck and great trochanter removed from the upper side. The part marked 1 is the lesser trochanter, and the part marked 2 shows the cancellous tissue of the spiral ridge, while the part marked 3 shows a few plates at the base of the lesser

FIG. II.

FIG. III.

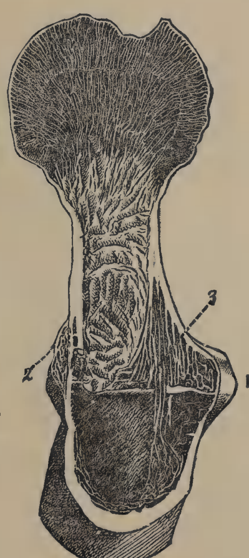

FIG. IV.

trochanter, fused into a bony plate having considerable firmness. In some specimens this subtrochanteric plate, or *spur*, of bone is absent, the entire structure being cancellous. The heavy compact tissue of the femoral brace will be seen in the neck of the bone.

3. The sections are made transversely to the femoral neck, and continued till they pass well through the trochanters. See figs. v, vi, vii, viii, ix. (a.) Fig. v represents a transverse section of the outer part of the femoral neck, intracapsular in front and extracapsular behind. The part marked 1 and 2 shows the femoral brace. The part marked 4 is the anterior compact tissue of the femoral neck, being thinner than the femoral brace. Part of the great trochanter is shown at 3. The cervical capsule may be seen at 5 and 5. (b.) Fig. vi represents a section from which the piece seen in fig. v was cut. The part marked 1 and 2 shows the femoral brace; figure 2 is at the extremity of the lesser trochanter; figure 4 shows the anterior compact tissue expanding into the greater trochanter; and figure 3 shows the posterior and upper part of the great trochanter; *while figure 5 shows the inner end of the subtrochanteric plate of compact tissue*. (c.) Fig. vii represents a section from which the piece seen in fig. vi was cut. The part marked 1 represents the femoral brace, which is seen to be continuous with the compact tissue of the femur in front, which, at 4, expands rapidly into cancellous tissue. At 3 is seen the compact tissue of the greater trochanter; *and figure 5 shows the subtrochanteric plate of compact tissue*, over which is seen the lesser trochanter, marked 2. (d.) Fig. viii represents a section from which the piece seen in fig. vii was cut. The part marked 1 represents the femoral brace, which is seen to be continuous with the compact tissue of the femur in front, which slowly expands into cancellous tissue. The lesser trochanter is marked 2, and the greater trochanter is

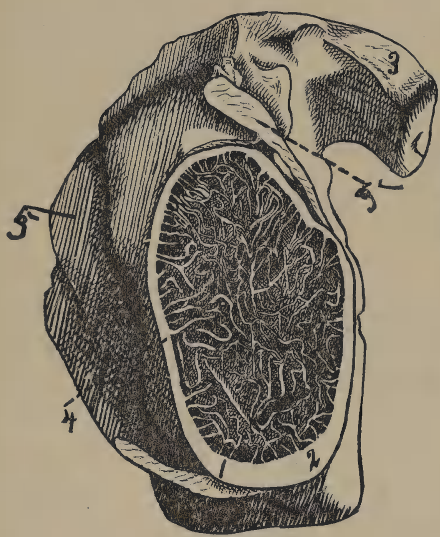

FIG. V.

FIG. VI.

FIG. VII.

FIG. VIII.

marked 3. *The subtrochanteric plate of compact tissue is shown at* 5. (e.) Fig. ix represents a section from which the piece seen in fig. viii was cut. The part marked 1 represents the femoral brace, and the part marked 4 is the anterior wall of the femur, while the part marked 2 is the out-set of the lesser trochanter. *At this point there is no subtrochanteric plate.*

FIG. IX.

The trochanteric pyramid is that eminence of bone found on the external aspect of the greater trochanter. In general the summit of the trochanteric pyramid is nearly over and external to the posterior wall of the femoral neck. This fact is readily seen by inspecting figs. ii, iii, iv, v, and vi. I find this condition to be a common one. In general the anterior surface of the upper end of the femur is, in the main, convex, while the posterior surface is concave. The practical importance of this condition will appear in due time.

The compact tissue of the anterior and posterior walls of the femoral neck expands into cancellous tissue at the base of the femoral head and at the trochanters. The femoral brace is firm and heavy at its base, while it is pointed and sharp at its junction with the femoral head. Hence it appears that the femoral neck is eminently fitted for being driven into the cancellous tissue of the femoral head or of the trochanters. Also it appears that a blow on the outer aspect of the upper end of the femur will be applied somewhat directly over the compact tissue of the posterior wall of the femoral neck, and will make the impaction greater behind than in front, so that in general the shaft of the femur will be out-rotated, when the base of the femoral neck is driven into the cancellous tissue of the trochanters. Also, it appears that the top of the femoral brace can be driven into the femoral head, when the femoral head will be rotated, or tilted, somewhat downward.

Let me draw especial attention to the relations of the compact and the cancellous tissues, as represented in figs. vii, viii, and ix. Figure 1 is on the heaviest part of the compact tissue, and from this point in opposite directions the compact tissue gradually becomes thinner, as it gives off the plates of cancellous tissue, not only in front but also behind. The compact tissue is actually expanded into the cancellous tissue,—only in some cases a few plates of cancellous tissue are given off in company, as already described, constituting quite a firm plate of bone. A blow on the femoral pyramid, as in falling on the trochanter, is applied almost directly over this plate of bone, when it exists at the base of the lower trochanter, so that the trochanteric pyramid is penetrated by this subtrochanteric plate of bone.

In the next place I desire to present for your consideration some specimens of fracture of the femoral neck that are the property of the Long Island College Hospital.

Specimen No. 1 is represented in Fig. x. It is the right femur. A longitudinal section of the upper end has been made and the anterior surface of the posterior half is shown. The femoral brace was broken at *c*, and driven into the cancellous tissue as far as *d*—over an inch. *There is firm bony union at c.* The point *c* was probably near the insertion of the capsule before the fracture took place. The femoral head is bent downward, indicating that there was a partial fracture of the upper part of the femoral neck. This is now shown by some shortening. There was out-rotation of the shaft of the femur, and the base of the femoral neck was tilted forward.

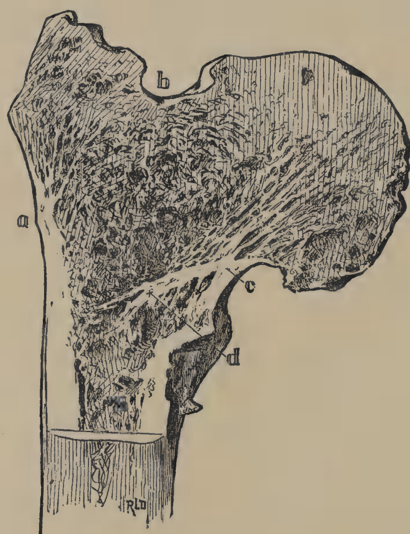

FIG. X.

Specimen No. 2 is represented in fig. xi. It is the right femur. A longitudinal section of the upper end has been made; and the anterior surface of the posterior half is shown. The base of the femoral brace has been broken at *c* and slightly impacted. The femoral brace was broken

at *e* and *d*, and with the under part of the femoral head driven outward and upward. The cancellous tissue was crushed and impacted. The upper wall of the femoral neck was broken at *a*, and driven about one fourth of an inch into the cancellous tissue. The head and neck of the bone have been carried outward and upward. There is bony union at *a*, and partial bony union at *c*; both of these points were near the original insertion of the capsule. At *e* there is new compact bone. At *d* there is new compact bone which comes up to the surface of extensive cancellous bone formed outside of the femoral brace. It may be remarked that this new cancellous bone—in the form of provisional callus—appears to be permanent. It will be seen that permanent cancellous bone has formed outside of the compact bone at *b*, on the outer side of the femur. It is difficult to prove that the femoral brace

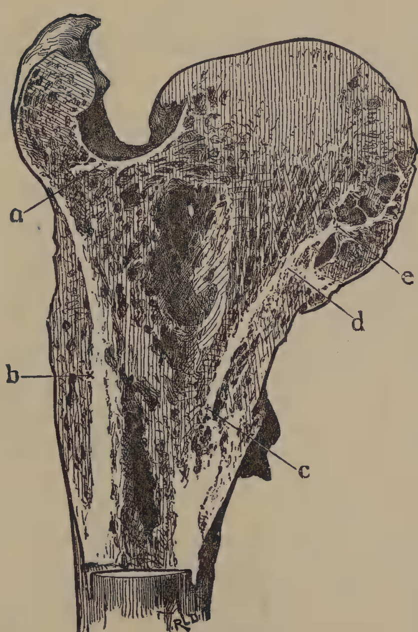

FIG. XI.

at *d* and *e* was broken; and yet, in my opinion, it must have been carried outward and upward at the upper end with a crushed femoral head—and that would be equivalent at least to an incomplete fracture. *And these points are inside the capsule of the hip-joint.*

In both specimens, figs. x and xi, there has been the actual formation of new bone, as well as the absorption of old bone, *and in specimen fig. xi, there has been a large quantity of new bone formed inside the hip-capsule.* And that new-formed bone is cancellous tissue, but not for that reason any the less bone. In fact, the union of fragments by cancellous bone is just as much bony union as the union of fragments by compact bone.

Specimen No. 3 is represented in figs. xii and xiii. It is the upper end of the right femur. A longitudinal section of the specimen has been made. The anterior half is shown in fig. xii. The posterior half is shown in fig. xiii. The cancellous tissue *f*, *f*, fig. xii, contains an abundance of fat. The fat has been removed from the cancellous tissue of the posterior half, as seen in fig. xiii.

The following points may now be noted:

1. *The top of the femoral brace has been broken from the femoral head, and driven into the femoral head one inch, and carried upward one inch,—an impaction of the femoral brace into the femoral head,—thus shortening the limb one inch.*

2. The cancellous tissue of the femoral neck above the femoral brace has been *comminuted and impacted.* Three pieces of bone can be seen on inspection, part of one piece being lost, as seen by fig. xiii.

3. Some of the cancellous bone of the femoral head, around the femoral brace, has been absorbed; the space left is about one eighth of an inch wide.

4. The femoral brace *c*, *d*, as represented in figs. xii and xiii, has not been materially changed, and is firm and strong.

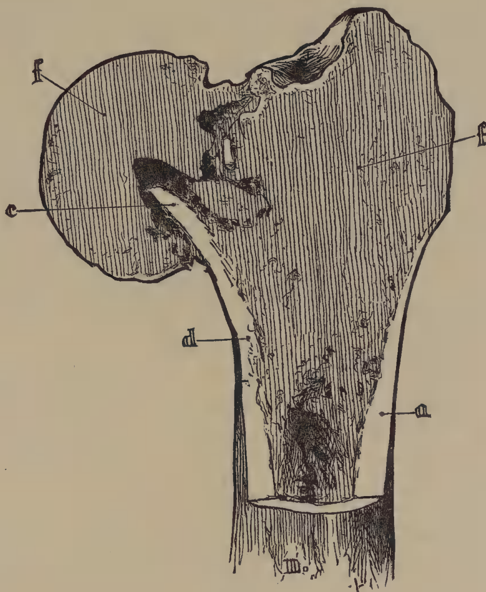

FIG. XII.

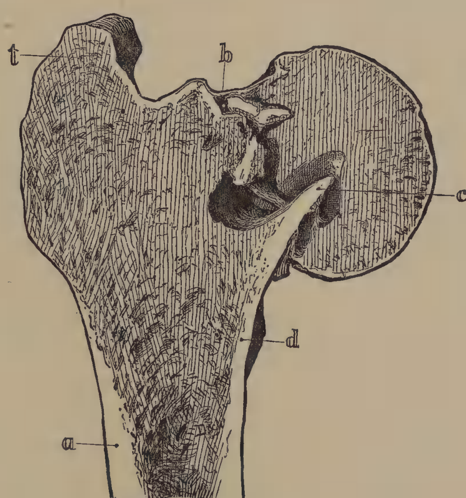

FIG. XIII.

5. The form of the trochanteric region has not been changed. The cancellous tissue contains more fat than normal.

6. From the anterior intertrochanteric ridge there has grown up an osteophyte one inch and three fourths in length, one half inch in width, and five eighths of one inch in height. The capsule is between the osteophyte and the head of the bone.

7. Posteriorly the capsule is inserted about the middle of the femoral neck, as it exists at present.

8. The femoral head has been turned downward till its transverse diameter is nearly parallel with the long axis of the shaft of the femur.

9. Before section of the bone there was no mobility at the seat of fracture—the impaction and the thickened capsule affording firm support—so that the patient must have had a useful limb. After section of the bone there was some mobility at the seat of fracture, showing that there was no osseous union.

10. This specimen was taken from the dissecting-room, and has no clinical history. It must have shown about one inch of actual shortening of the lower limb at the time of the injury, and could not have shortened very much subsequently. As there is no injury to the trochanters, and as there is fatty degeneration of bone and osteoporosis, the breaking force could not have been very great. And it may have been very difficult to make an exact diagnosis, yet it might have been conjectured that the femoral brace had been driven into the femoral head. In fine, it was no doubt possible for the patient to continue to get up and walk about to some extent, as if there had been only a contusion of the hip.

11. This was an *intracapsular fracture*. In time, if the patient had lived, the femoral brace and the neck would

have been absorbed, so that the femoral head would have come against the upper end of the shaft, when it could have been alleged, without fear of contradiction, that there had been an extracapsular fracture so-called of the neck of the femur, and that the entire femoral neck had been absorbed.

12. In a fracture of the kind under consideration, when the osteoporosis does not interfere with the formation of bone-scar, it is easy to see that good union might occur, either of *bone-scar*, or of scar tissue.

13. An attempt to obtain crepitus in this case must have failed, because the impaction of the femoral brace would have caused the femoral head to follow the forced motions of the outer fragment. And violent attempts to obtain crepitus, with or without an anæsthetic, would have produced material additional damage to the injured parts. And it can be seen that proper measurements, keeping in mind the subject of asymmetry, would enable the surgeon to make a diagnosis of fracture of the neck of the femur.

14. In fine, it may be remarked that, when the evidence is put in proper form, it will be found that an impaction of the femoral brace into the femoral head is not a very rare accident.

Specimen No. 4 is represented by fig. xiv, which is the posterior part of the upper end of the *right* femur. The compact tissue of the outer part of the femoral shaft is shown at *a*. The femoral brace is shown at *d* and *c*. The femoral brace is thick and strong at the upper part, and the summit of the femoral brace penetrates the cancellous tissue of the femoral head. *At the same time there is an angular displacement of the femoral head downward and outward about twenty degrees.* The angle of displacement is well marked at *b*, on the upper side of the femoral neck; and on the outside of the femoral neck all round is a ridge

of firm new bone, which has the appearance of callus. There is no abnormality in any other part of the upper end of this femur. About nine inches from the top of the shaft the femur has been broken and united with less displacement than there is in the femoral neck at *b*, *c*. This angular displacement of the femoral head may be explained on one of two assumptions: (1) The appearances may be the result of an abnormal growth in connection with abnormal or unusual pressure on the top of the femoral

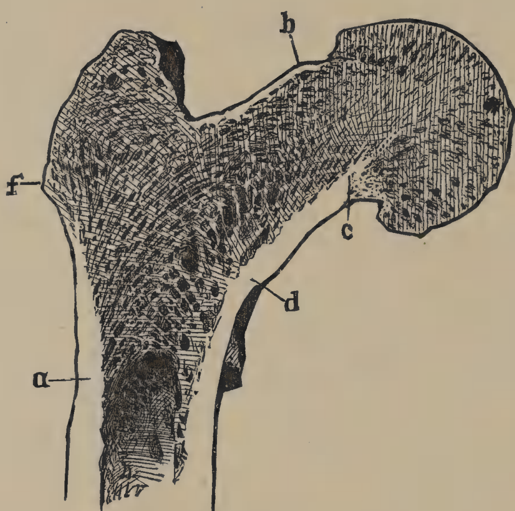

FIG. XIV.

head,—just as trees have their branches developed most on the side away from a prevailing wind. And this is of the nature of a traumatism persistently and slowly acting, —the cancellous tissue bending in an organic way, and the compact tissue of the femoral brace, being of an unyielding nature, penetrating the more yielding bone. (2.) Or the appearance may be the result of a sudden traumatism causing an incomplete fracture of the femoral neck, in which the femoral brace is detached from its normal

place and driven into the cancellous tissue, and the cancellous tissue is bent and partly broken; and subsequently bony union takes place within the femoral capsule, as indicated by the upper part of the femoral brace being thicker than normal, and by the firm ridge of new bone in the exact line where the femoral neck is supposed to be partially fractured. Now, as to causation, and as to pathogenesis, these two hypotheses are practically the same: if one explanation is admitted the other may be admitted as adequate. As for myself, I incline to the view that the traumatism must have been somewhat sudden; in fact, I can see no reasonable objection to the second hypothesis. And if that is so, then there has been an incomplete fracture of the femoral neck within the capsule and subsequent bony union. This specimen came from an old man in the dissecting-room of the Long Island College Hospital.

Specimen No. 5 is represented by fig. xv, which is the posterior part of the upper end of the *left* femur. This specimen and specimen No. 4 were taken from the same subject. And Dr. A. J. Dower, who gave me this specimen, says it came from an old man with gray hair.

The upper part of the femoral brace penetrates the femoral head about one half inch: *There is an angular displacement of the femoral head downward and outward of about twenty degrees.* The angle of displacement is well marked at *b*, on the upper part of the femoral neck. And on the outside of the femoral neck is a ridge of firm new bone, which has the appearance of callus. On the outer aspect of the upper end of the femur the compact tissue *a* goes upward at *e* into the cancellous tissue; and the appearance is that the entire cancellous tissue of the top of the femur has been carried outward and downward by the *slow* or the *sudden* application of force to the distance of about one half inch. And it is noteworthy that the dis-

tance of the point *f*, in each of these specimens, from the top of the femoral head, is exactly the same,—being four and one fourth inches. In one case—specimen No. 4—the head of the bone has been bent downward. In the other case—specimen No. 5—the head of the bone has been bent downward, and the entire cancellous tissue has been carried outward; the thin plates of cancellous tissue appear to be bent outward, and do not run upward and inward as in the normal condition.

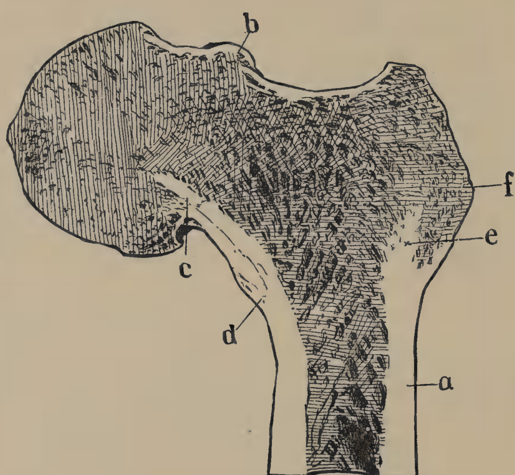

FIG. XV.

This abnormal position of the femoral head and the rest of the cancellous tissue of the upper end of the femur may be explained on one of two assumptions: (1) The appearances may be due to an abnormal growth in connection with abnormal pressure on the top of the femoral head partly downward and partly outward. The cancellous tissue has slowly given way at the top of the femoral brace and at the top of the compact tissue on the outer aspect of the femoral shaft. (2) Or the appearances may be due to a more sudden traumatism, causing an incomplete frac-

ture of the femoral neck as well as of the entire cancellous tissue of the upper end of the femoral shaft. The top of the femoral brace penetrates the femoral head, and the compact tissue of the outer aspect of the femoral shaft penetrates the cancellous tissue of the greater trochanter. As before stated, these two hypotheses are substantially the same: if one explanation is adequate, the other is also adequate. Yet I incline to the view that the traumatism was somewhat sudden.

In these two specimens—Nos. 4 and 5—the compact tissue is very firm and solid, and the cancellous tissue is entirely free from fat, being normal in every respect except one, and that is as to the distribution of its plates. (a) In specimen No. 4, the plates of cancellous tissue are quite normally distributed in every part but at the place of angular displacement. (b) In specimen No. 5, the plates of cancellous tissue can not be said to be normally distributed in any part, unless it be in the middle of the femoral neck.

In these two specimens I think we may exclude both osteoporosis and fatty degeneration. To be sure, old age and mechanical pressure were present, and there are certain notable deformities present, even greater deformities than are found after a fracture of the shaft of the femur, to which one of the specimens belongs. So that the deformity is sufficient to be the result of a traumatism. But it may be said that there is no mark of callus *in* the cancellous tissue. In answer to this there is no mark of callus in the compact tissue of the femoral shaft of specimen No. 4, where there has been a fracture.

In fine, let me remark that there are two other ways by which these abnormalities are explained: (1) Some one says *they grew so.* (2) Some one else says *they are cases of interstitial absorption.* Let me reply. Shall we ignore the

dynamic relations of these two bones, and only admit some of the elements of the practical problem? And shall we say that good union—even bony union—may not take place sometimes within the femoral capsule, when accidental or surgical interference does not prevent such a desirable result?

EDITORIAL DEPARTMENT.

"CHEMICAL RESTRAINT" IN THE MANAGEMENT OF THE INSANE.

An impression prevails among the medical profession in the United States that in the British asylums for the insane the necessity for restraint by mechanical appliances, such as camisoles, muffs, straps, crib-bedsteads, etc., is obviated by the free use of stupefying drugs. The prevalence of the opinion is easily accounted for. In the United States the subject of the management of the insane, as well as the special study of insanity, has been left by the profession, in the main, to the superintendents of insane asylums. So when these gentlemen, in the annual meetings of their association and in their annual reports, have persistently asserted this substitution of "chemical" for mechanical restraint in British asylums, the medical profession have generally accepted the statement as a fact.

It was a borrowed assertion at the start, but by long-continued iteration it has become an article of faith with the superintendents of American asylums. It had its origin among the British opponents of "non-restraint" more than thirty years ago. These men, like their American brethren of the present day, believed that the only recourse for dealing with madmen was some form of restraint. With the disuse of mechanical restraint they could conceive of no alternative but resort to prostrating remedies, or of a superior physical force at the hands of muscular attendants. In a letter received from one of the oldest of our superintendents, under date

of September 12th, in defending the use of restraining apparatus, he says:

"I believe that in many cases such means of control and care are alike more effective and humane than the opposite practice of attempting to control reckless, excited, and violent cases by the superior strength of one or several attendants, as the practice of some is, or of 'laying them out' by the use of strong drugs, as is said to be the resort of others."

Dr. Gray, the editor of the *American Journal of Insanity*, on his return from a late visit to Europe, informed his brethren of the New York State Medical Society that some of the English superintendents "used practically chemical or medicinal restraint, which, in the end, would prove more injurious than mechanical restraint."

As to the former alternative, it may be said that while one can hardly pass through any of our state asylums without seeing the various forms of mechanical restraint more or less used, yet I may say, after a quite extensive personal observation of the refractory wards of British asylums, that I have never happened to witness any patients struggling in the hands of attendants. And I also observed that the female patients were controlled by female attendants not of conspicuously muscular proportions. As to the other alternative, the statistics hereafter to be presented will throw some light.

Happily the experience of the British alienists has not confirmed these and other theoretical objections to the principle of non-restraint. For in the presidential address of Dr. D. Hack Tuke before the British Medico-Psychological Association, in August last, I find the following sentence: "No one will call in question the statement, as a historical fact, that the commissioners of lunacy and the medical superintendents of asylums in this country are, with few exceptions, in favor of 'non-restraint.'"

In the matter of professional communion the Straits of Dover is as wide as the Atlantic, and so we find that some of the French alienists, unaffected by the triumph of non-restraint in Great Britain, are urging the same objections to the doctrine common

in this country, and especially the allegation that chemical restraint is the substitute for mechanical restraint in the British asylums.

It becomes, therefore, a question of some importance to determine whether the statement is true. It occurred to me that I could, by some comparative statistics from the asylum registers of Great Britain and the United States, settle the question to the satisfaction of the medical profession in this country.

It is hardly necessary to say that there is a legitimate use for chloral and the narcotic remedies in the treatment of insanity as in other diseases. Some of the more commonly manifested symptoms of the disease obviously indicate their use. Nevertheless here, as in the domain of medicine generally, drugs are not to be given when other remedial agencies will avail. But I think that it will appear from the accompanying statistics that in some asylums soporifics and sedatives are administered so continuously and in view of such indications as to warrant the term "chemical restraint."

That chloral is given empirically no one can doubt. It is also evident that pathological considerations do not determine, always if ever, in the employment of hyoscyamia. At all events, we find one superintendent of an asylum who has used it largely, internally and by hypodermic injection, commending its use in acute mania, chronic paroxysmal mania, melancholia, and paresis, besides finding it useful in hysteria and chorea. As he puts it, "The cases of mania in which it was administered may be divided into those who are maniacal, raving, noisy, incoherent, and opposing necessary care, and destructive of clothing; second, such as have occasional periods of maniacal excitement; and third, such as are uneasy, talkative, restless, and sleepless. The cases of melancholia may be divided into three classes; such as had periods of frenzy, sometimes endangering life; such as persistently and determinately resisted care and food under delusions; and such as would wear themselves out from restlessness and constant motion."

The *modus operandi* of a remedy is supposed to determine its

application, and he therefore suggests the following principle for the guidance of others: "I am inclined to think that it produces an effect upon the cerebral nerve tissue of a beneficent character, quieting the irritable and excited brain quite as markedly as preparations of opium, in their influence on nerve tissue, will relieve pain."

"Dr. Kempster, of the Wisconsin Asylum," according to the statement of Dr. Squibb, who is engaged in the manufacture of hyoscyamia, "has used this remedy quite largely, and considers it as a most admirable substitute for physical restraint. He says that with the proper use of hyoscyamia he thinks it may never be necessary to use the straight-jacket or other means of restraint, and that the maniacal patients who cannot be controlled by its use he believes are very rare. He uses it hypodermically."

In this case it is not an improper use of language to speak of it as chemical restraint.

Some fifteen months after the introduction of chloral at the Utica Asylum Dr. Andrews described the mode of its use.

"The whole amount used is 90 lbs., which has been prescribed in 370 cases, as follows:

FORM OF DISEASE.	MEN.	WOMEN.	TOTAL.
Mania . . .	69	119	188
Melancholia . .	30	59	89
Dementia . .	18	50	68
Paresis . . .	12	1	13
Epilepsy . .	2	2	4
Employés . . .	3	5	8
	134	236	370"

It is further related that 15 of the number took it nightly, on an average, for some 200 successive days. The average dose employed 30 grains. It can hardly be questioned, then, that in that particular institution this remedy was used in a somewhat routine way.

Some five years since a fatal accident happened in a Western

asylum. An investigation followed by the Board of State Charities. In their report to the Governor occurs the following paragraph: "The use of chloral hydrate to produce sleep at night, common, as we are informed, in the majority of hospitals, is carried to a considerable extent at this asylum. The night-list of medicines administered shows that about sixty patients (ten per cent.), on an average, take chloral every night, the average dose being from 30 to 35 grains, in combination with whiskey, opium, or fluid extract of hyoscyamus."

It may be well at the outset to say that it would be easy to furnish competent general statements from British authorities in denial of the assertion that "chemical restraint" is the substitute for mechanical restraint in British asylums. One will suffice.

The Lunacy Commissioners of Scotland remark in their annual report for 1877: "Mechanical restraints and seclusion are probably as little used in the treatment of the insane in the asylums of Scotland as in those of any part of the world." And again, "Stimulants appear to be decreasingly consumed in Scotch asylums. * * * Even more than in the case of stimulants, the use of narcotics appears to be diminishing. In some large asylums sleeping draughts are rarely given. Increasing attention, however, is bestowed on all those arrangements which tend to secure sound and refreshing natural sleep."

But to come to comparative statistics, it may be remarked, then, first, in a general way, that the cost of medical supplies in British asylums for the insane is very much less than in those of the United States. The average annual cost, per patient, for such supplies in Great Britain is about one dollar. (See Lunacy Reports for 1880.) The cost in American asylums, from two to six times as much.

Secondly, to attain more precise results, the following circular was sent to the superintendents of some 20 British asylums:

"Dear Sir:

"It is alleged in this country by the opponents of the doctrine of 'Non-Restraint' that in the British asylums, where restraining

apparatus is least used, resort is had in large degree to the use of the so-called chemical restraints. I have prepared the accompanying circular of questions, to be sent to a dozen or more British asylums, where restraint is least used, that I may learn the extent to which sedatives and narcotics are used as substitutes for mechanical restraint.

"Will you kindly fill out the enclosed blank and return it to me."

"Name of Institution . . . . Number of Patients . . . . Males . . . . Females . . . .

"Average number of patients to whom chloral is administered each day . . . .

"Average number of patients to whom hyoscyamia or other narcotic is administered to allay excitement . . . .

"Number of instances of seclusion for a month past . . . .

"Number of occasions for the use of mechanical restraint for a month past . . . .

"Remarks."

I am now able to give the returns from 15 British asylums. I have also, by correspondence, obtained similar statistics from some American institutions. These are embodied in the following tables.

With reference to the first two tables, namely, those relating to British and Canadian asylums, it should be remarked that the statistics were taken from the registers of the several asylums for the month preceding the receipt of my request.

It is hardly necessary to say that these statistics relate to institutions that are recognized as among the best in Great Britain, that the medical superintendents who are in charge of them are conspicuous for ability and success, and, further, that the ratio of recoveries in these asylums will compare favorably with that of similar institutions in the United States.

To these tables should be appended some of the remarks that have accompanied the returned circulars.

Thus, the patient restrained at West Riding was so restrained for surgical reasons. Patients there, especially epileptics, are frequently kept in bed, for excitement, the door not being fastened. Only one patient has been restrained since 1877.

TABLE NO. I.—BRITISH ASYLUMS.

Name.	No.	Number of Patients.	Monthly occasions of restraint.	Monthly number restrained.	Monthly occasions of seclusion.	Monthly number secluded.	Average number to whom chloral is daily administered.	Average number to whom hyoscyamia or other narcotic is administered to allay excitement.
West Riding Asylum . . .	1	1,410	1	1	2	2	31	38
County Asylum, Chester . . .	2	533	none	none	none	none	none	4 Morphia used occasionally
Hull Borough Asylum . . .	3	163	"	"	"	"	1	2
Montrose Royal Lunatic Asylum	4	485	"	"	19	1	3	1
Brookwood Asylum, Surrey .	5	1,050	"	"	none	none	10	1
East Riding Asylum	6	286	"	"	"	"	½	none
Hanwell Asylum	7	750	"	"	"	"	none	"
Burntwood Asylum, Litchfield	8	600	"	"	"	"	"	"
Royal Edinbur'h Asylum. . . .	9	832	"	"	20	"	1	"
North Riding Asylum . . .	10	546	2	1	3	"	7	9
Royal Asylum, Gartnavel . .	11	483	none	none	none	none	6	14
Richmond Dist. Asylum, Dublin	12	1,013	"	"	3	1	11	2
Dr. W. C. Hill's, Norfolk County	13	620	"	"	none	none	none	20
Kent Co., Asylum	14	1,200	"	"	"	"	"	none
Woodilee (near Glasgow . . .	15	448	"	"	"	"	"	"
		10,419	3	2	47	4	70½	91

TABLE NO 2.—CANADIAN ASYLUMS.

Name.	No.	Number of Patients.	Monthly occasions of restraint	Number restrained.	Monthly occasions of seclusion.	Number secluded.	Average number to whom chloral is daily administered.	Average number to whom hyoscyamia or other narcotic is administered to allay excitement.
Nova Scotia Hospital for Insane	1	380	117	11	17	5	none	none
London, Ont., Asy'm for I'sane	2	851	61	8	13	9	"	"
Toronto, Ont., Asy'm for I'sane	3	673	10	3	4	2	2	3
Hamilton, Ont., Asy'm for I'sane	4	537	6	—	4	—	5	5
Kingston, Ont., Asy'm for I'sane	5	430	4	—	13	—	occasional	occasional
		2,871	198		51			

TABLE NO 3.—ASYLUMS IN UNITED STATES.[1]

Name of Asylum.	No.	Number of pati'nts.	Monthly occasions of restraint.	Number restrained.	Monthly occasions of seclusion.	Number secluded.	Average number to whom chloral is administered daily.	Average number to whom hyoscyamia or other narcotic is administered to allay excitement.
Northern Hospital, Wisconsin	1	541	48	—	1	1	24	[illegible] daily
Cook County Asylum, Illinois	2	440	480	—	60	—	33	—
Kings County Asylum, N. Y.	3	868	none	none	—	8 daily	$5\frac{1}{2}$	6 a month
Worcester Hospital, Mass.	4	594	69	—	71	—	22	no record
Retreat for Insane, Hartford, Conn.	5	121	2	—	2	—	4	1 daily
Willard Asylum, New York	6	1,727	—	6 daily	7	—	27	10 "
Athens Asylum, Ohio	7	586	none	none	116	58	20	none
Longview Asylum, Ohio	8	661	8	—	11	—	8	1 daily
Dayton Asylum, Ohio	9	591	118	6 daily	309	—	29	—
Northern Asylum, Elgin, Ill.	10	526	483	—	25	—	26	5 daily
Insane Criminal Asy'um, N.Y.	11	131	1	1	5	—	$1\frac{1}{3}$	5 a month
Middletown, Connecticut	12	582	16	3	43	15	21	1 daily
Minn. Hospital for Insane	13	530	67	43	24	17	6	[illegible]
Southern Asylum, Anna, Ill.	14	486	350	—	129	—	12	3
Eastern Illinois Asylum	15	175	12	—	22	—	—	3
Homœopathic, New York	16	244	5	3 daily	—	—	none	none
Central Hospital, Illinois	17	641	483	32	[illegible]	—	73	48
Western Asylum, Kentucky	18	473	16	—	12	—	9	8
Hudson River Hospital	19	250	—	6	none	—	29	12
State Insane Hospital, Wis.	20	548	2,547	—	13	—	4	10
Danvers, Massachusetts	21	643	138	—	161	—	5	3
Northampton, Massachusetts	22	471	25	10	26	—	none	none
S. Lunatic Hospital, Harrisburg, Pa.	23	353	3	—	21	—	18	3
Taunton Lunatic Hospital	24	574	14	—	6	—	20	—
N. J. S. Lunatic Asylum	25	586	180	—	150	—	118	none
Newburg, Ohio	26	625	24	—	38	—	47	21
(see postscript)		13,967			1,254		561	139

[1] This table is incomplete in some of its columns because the reports upon which it is based were imperfect.

Dr. Major also remarks: "I believe it to be a great mistake to consider that with us non-restraint depends in any degree upon our use of sedatives. Were sedatives taken from us entirely I am sure we should not use more than we do now. Here, also, I have cases who have *most destructive tendencies and habits*, and who, I feel sure, if restraint were in use would be restrained; but they are not restrained and are not habitually on sedatives. I still believe that in *rare* instances restraint (other than surgical) is of advantage to the patient, and, therefore, should be resorted to; but I think those cases so rare as to be quite an event in procedure."

The case of seclusion at the Montrose Asylum was a homicidal epileptic, who, after a series of fits, voluntarily remains in bed; at other times works on the farm on parole.

Dr. Whitcombe, of the East Riding Asylum, remarks: "At the present time not a single patient is under treatment to allay excitement. The chief means used here are employment, out-door exercises, and in- and out-door amusemements. Restraint and seclusion are rarely, if ever, needed. I look upon chemical as one of the most pernicious forms of restraint,"

Dr. Brushfield, of the Brookwood Asylum, says "that mechanical restraint has not been used in the asylum since its opening in 1867. Seclusion (that is, shutting up a patient by himself) has not been practised since the year 1875. The numbers given are beyond the usual average; and such remedies are never used continuously with any patient for any period."

Dr. Rayner, of the male department of Hanwell Asylum, adds: "In the nine years I have been here I have never used mechanical restraint, although I should not hesitate to do so if the necessity arose. I never use sedatives to allay excitement, and narcotics to procure sleep very rarely; no patient has sleeping draughts as a habit, and probably not more than two or three such draughts are given in a month. My rule is, 'Better no sleep than a stupor from drugs,' of whose action we only know that they gravely affect the processes of nutritional repair; that the most protracted cases of mania are those in which narcotics have been used; the most in-

tractable cases of insanity, those which have been most freely treated with sedatives and narcotics."

Dr. Davis, of the Burntwood Asylum, says: "I have just left the asylum, but during the 17 years that I was there I had no recourse to seclusion or restraint. I always found that plenty of food and extras, such as stimulants in arrow-root, quieted the most turbulent cases."

Dr. Nicholson, of the North Riding Asylum, says "that the two occasions for restraint mentioned were in the case of one individual for surgical reasons. Hyoscyamia is only given in two cases at present, the principal other sedatives being opium and its preparations."

Dr. Yellowlees, of the Gartnavel Asylum, referring to his figures, says: "Chloral is given at bedtime and usually with bromide of potassium. It is very rarely given by day, and at night only if required. Two (2) melancholics are taking regular doses of liquid extract of opium, and about 12 patients, including epileptics, are taking bromide of potassium regularly. This is the whole sedative drug treatment for the month. Hyoscyamine has never been used here, as I am satisfied it is dangerous, and greatly doubt if it is really beneficial. The two patients secluded were each secluded on two occasions. One of them is an epileptic. Last month there was but one patient in seclusion, and only on two occasions, for epileptic mania. I do not hesitate about using restraint if I think it necessary for the patient's welfare, but have had no case requiring it for a long time. Such a case may not occur once in a whole year. Of course I exclude cases where some surgical necessity may demand it. I do not consider the use of padded gloves, enveloping the whole hand, to be "restraint" at all, and occasionally employ them in cases of determined suicidal attempts or of extreme destructive violence, but only with express medical sanction. * * * * * Mechanical restraint tends in the vast majority of cases to the injury of the patient instead of to his benefit, and therefore—*and only therefore*—it should be dispensed with as far as possible,—which means, practically, that it is all but completely disused. I hold a similar

opinion about the so-called 'chemical restraint,' as my practice proves."

Dr. Davies, of the Kent County Asylum, said in a letter to the *Journal of Mental Science*, Jan., 1881: "Chemical restraint has long since ceased to be practised here. I did not make the change suddenly; it has been a gradual transition. I used to give large doses of morphia, chloral, etc.; then less, and now none."

Dr. Hill, of the Norfolk Asylum, "while not an advocate for the routine use of sedative drugs, thinks his practice of administering them to about three per cent. of his patients beneficial."

Dr. Merson, of the Kingston Asylum, says: "The returns I send you represent perhaps more than the usual average of patients taking medicine to allay excitement. Chloral I never use to allay excitement, except in the case of epileptics subject to outbursts of fury before or after fits, and in these cases its action is simply marvellous. I never give it for prolonged periods in cases of chronic excitement. My experience here is of course limited to small numbers, but so far as it goes I am inclined to think that, as with mechanical restraint so with chemical restraint, the less they are used the less need we shall have to use them."

Dr. Rutherford, of the Woodilee Asylum, says in his last annual report, after describing the thoroughness of his system of occupation of patients: "This full employment of the patients renders it possible to give greatly extended liberty, and to do away with all remaining forms of mechanical or chemical restraint, such as walled courts, locked doors, stimulants, narcotics, and sedatives."

In the Montrose Asylum for the whole year 1877, with 549 patients, only three men and twenty-two women had draughts given to induce sleep, most of them only occasionally, some only once. In only four instances were the draughts given continuously.

In the English as in the American asylums the Irish are regarded as the most turbulent patients; yet it will be seen by the returns from the Richmond Asylum, Dublin, that with 1,013 patients there was no mechanical restraint, and only one patient

secluded, on three occasions and for a total period of four days and four hours.

Dr. Lalor also kindly sends me his record of seclusion for the prior six months. From this I learn that thirteen patients were secluded on one occasion each, one patient on two occasions, and one on five. The total aggregate period of seclusion of the fifteen was 205 hours.

Accompanying the Canadian tables were the following remarks :

"The cases of seclusion at the Nova Scotia Asylum were for brief periods, usually from one to three hours. Under restraint: two male patients muffed, nightly, and two females, one for four nights and the other for twelve, constitute the most of the occasions."

Dr. Bucke, of the London Asylum writes, that the average duration of each instance of seclusion was less than two hours ; of each instance of restraint about nine hours. He adds : "I use no sedatives here, and no *alcohol* in any form. I find that my patients rest better at night and need sedatives less, since I ceased to use alcohol. I never give medicine except for bodily ailments. Restraint of all kinds has been much reduced at this asylum during the last few years. I hope to do without it altogether, after a time."

Dr. Clarke, of the Toronto Asylum writes that to no patients is either chloral or any other drug given habitually or continuously. In the male department only three have been restrained in thirty-two months.

Dr. Wallace, of the Hamilton Asylum writes that the figures for the past month, are in all respects higher than usual ; chloral is never given except in cases of extreme excitement. Merck's hyoscyamine is used, and is in many respects superior to chloral, though the after-effects are more disagreeable. The only forms of restraint used are the camisole, muff, and restraint-bed. In cases of extreme excitement and continued insomnia the restraint-bed is used with the best results to the patient. It enforces rest without the use of sedatives, which must always be more or less injurious when frequently repeated. The patient cannot injure him-

self or others, and the position almost always induces sleep and prevents fatal exhaustion in violent maniacal cases. Muffs are sometimes used, but the camisole preferred, being less uncomfortable and quite as effective. Alcohol has not been used in any form unless in tinctures for the past two years.

Dr. Metcalf, of the Kingston Asylum writes: "None of our patients regularly or constantly receive sedatives or narcotics. We administer an occasional dose whenever we think benefit will be derived. Sometimes we give half a dozen doses to the same patient, but rarely more than one or two consecutively. Seclusion simply means putting the patient, for as short time as possible, into an ordinary single bedroom. We use for restraint the leather muff, or simply the wristlets belonging to the muff."

Remarks accompanying and explanatory of the statistics of asylums in the United States:

In the table relating to asylums in the United States I have purposely omitted the names, designating them only by numbers to avoid offence. Two of the asylums, Nos. 2 and 3, are county institutions; the others, with one exception, state institutions.

In some of the States where there were several asylums my application for the statistics was made through the Boards of State Charities. In others it was made direct to the medical superintendents. In a few cases the record was made for the month succeeding the date of application, and, therefore, possibly may not represent the average monthly record for the year. In interpreting the term "occasions" of restraint or seclusion, it may be well to state that the reporter, as a rule, has called a week or month of continuous restraint as seven or thirty occasions, as the case may be.

The superintendent of No. 1 remarks that the numbers do not refer to the same individuals; in other words, that the patients are not necessarily taking the remedies continuously. Of the patients in No. 4 it is remarked that "fifteen are convicts." The total number of hours that patients were secluded was 1,601. This number was swelled by the fact that three persons were in seclusion for the whole twenty-four hours during the entire month.

The superintendent of No. 5 says: "The cases of mechanical restraint were two females, and only for short periods; and consisted of a linen waist with closed sleeves. In one case the patient was persistently suicidal, and in the other destructive of clothing."

No. 6 is doubtless improperly stated in the table. The return states that, on an average, three by day and three by night are restrained for suicidal propensities.

The return from No. 8, under instances of seclusion, reads: six regularly; eleven, total.

The return from No. 9 states that "the number 29 includes all who take any kind of sleeping draught"; also, "average number of instances of seclusion for each day of the month, $2\frac{2}{3}$, males, and $7\frac{3}{5}$, females; of restraint for each day, males, two; females, four."

The superintendent of No. 10 remarks—and the remark will doubtless apply to other American asylums—"that during the summer months there is greater need of restraint on account of greater irritability of patients."

The superintendent of No. 11 reports that only three of the cases secluded were on account of mental excitement.

The superintendent of No. 13 writes: "I wish to say that if the number under restraint appears large, it is made so, to a considerable extent, on account of our crowded condition. It is not quite proper to compare our list with a hospital where each patient can have a separate room, when we are obliged to keep two and three in *single* rooms and apply restraint to prevent assaults and homicides."

The superintendent of No. 15 gives the average duration of the periods of seclusion as nine hours.

The report of No. 16 in the table is doubtless improperly stated. The return reads: "Restraint of a mild form is used on between *one and two per cent.*, on the average."

The superintendent of No. 17 gives the following explanation of his use of restraint:

4	wore wristlets	during	month,	for violence.
4	" "	"	the day, for the month,	for violence.
2	" "	"	21 days	" "
2	" muff	"	14 "	for general destructiveness.
3	" "	"	21 "	" " " " of bedding.
1	" "	"	26 nights,	violence to self.
1	" "	"	8 days	" " and others.
1	" "	"	19 "	" " "
1	" "	"	$3\frac{1}{2}$ "	destructiveness.
2	" "	"	1 day	"
3	wore camisole	"	7 days	"
1	" "	"	2 "	"
2	" "	"	1 day	"
1	" "	"	$1\frac{1}{2}$ "	"

1 confined to bed to economize strength.

1 restrained to bed 3 nights.

The superintendent of No. 20 remarks: "The restraint has been for short periods, averaging one hour and a half. The occasions for restraint are often, with a few patients, four or five times daily of one hour each, during periods when they cannot be watched by attendants, and generally consist of a belt passed around the patient and through metal loop on chair. The construction of the building and its crowded condition renders seclusion almost impossible. The large number (40 to 50) in a ward makes mechanical restraint necessary in a much greater number of cases than would otherwise be useful."

The asylum, No. 20, is in the same state with No. 1. The State official whose duty it is to inspect these institutions, and who sends me these returns, writes as follows: "Notwithstanding the large discrepancy which appears on the face of the answers to the questions, I am satisfied, from a somewhat careful inquiry, that if any, there is no substantial difference in the amount of restraint practised in our hospitals. The difference is in the manner of reporting."

The superintendent of No. 25 gives chloral only as a hypnotic; the dose usually given 15 grains, seldom more. Narcotics are never given to allay excitement. No bad effects have resulted

from his mode of administering chloral. No cases are ever kept under constant seclusion or restraint. Restraint not applied, except by the authority of the physician. Patients from the state prison are sent to this asylum,—many of them homicidal cases ; hence, in part, the amount of mechanical restraint.

Some half a dozen of the superintendents of asylums in the United States have failed to respond to my inquiries, evidently unwilling to furnish the desired statistics. With reference to one of these, I learn from an authentic source that about ten per cent. of the patients are daily taking either chloral, hyoscyamia, or a combination of the two remedies.

Dr. Tuke, in the inaugural address from which I have already quoted, in comparing the old system of management of the insane with the new, remarks : "The old system desired secrecy ; the new is not afraid of publicity." It is evident that some of the institutions of the United States have not fully come out from under the influence of the older system.

My space would not allow me to give all the explanatory remarks accompanying the statistics of the several asylums. I have meant to give enough to avoid doing injustice to any. I may, therefore, now proceed to make a few comments on the above tables.

First, the use of sedatives and narcotics, the so-called "chemical restraint," is not the substitute or alternative for mechanical restraint either in British asylums or in the two or three American asylums where the principle of non-restraint has been lately on trial. On the contrary, the general rule seems to be : the more mechanical restraint, the more chemical restraint.

On reflection, this need not surprise any one. For if, as one American superintendent states it, "rest is vital to successful treatment of acute mania," mechanical restraint will not suffice, as it merely limits the range of muscular action, neither fully controlling the patient's efforts nor quieting the violent and exhausting action of his vocal organs. Till some ingenious superintendent shall invent a protective gag and still more efficient appliances of restraint, resort must be had to sedative drugs to secure the

vital rest. And so one superintendent writes that the narcotics he gives are not as substitutes for restraint, but in some cases associated with restraining apparatus.

Secondly, it appears that the British superintendents who have furnished these statistics not only do not regard such remedies as proper substitutes for mechanical restraint, but rather look upon their general use as unnecessary and even pernicious. Some of them even assert that such use protracts or perhaps prevents the recovery of the patients.

Thirdly, it will be seen from the opinions of the British superintendents, given in connection with the tables, that non-restraint, as held and practised by them, is no inflexible dogma. It is simply the practical disuse of restraining apparatus, because they have found by experience that other means and resources are better for the patient, except in very rare instances, in which event they would unhesitatingly accept the alternative. Of course, it will be seen that, besides the actual condition of the patient, the knowledge, tact, and skill of the physician will be factors in determining the application of the *dernier ressort.*

As has been already stated, the purpose of the present inquiry was merely to bring out the facts as to the comparative use of chemical restraint in British and American asylums. Incidentally another has been served. It is this. Although during the last four or five years there has been a great diminution in the use of restraining apparatus in the insane asylums of the United States, yet it is obvious from the table that mechanical restraint is now used in some to a degree that will surprise most British alienists. Some of their number have visited a few of our institutions when in this country. They have been told by the superintendents that little resort was had to restraining apparatus, and with the known non-restraint opinions of such visitors, such apparatus has, not unnaturally, been kept out of sight and out of use for the time being. These gentlemen have gone away deceived, as Dr. Bucknill was, as to the amount of restraint used. He spent several days at the Utica Asylum, and also travelled with Dr. Gray, its superintendent; and yet he wrote of his visit in his Notes on American

Asylums: "*That he saw none in restraint or seclusion at Utica, and that Dr. Gray differed from his American brethren in not using restraint.*"

It will not be out of place, in connection with these tables, to give a summary of the facts relating to the use of restraint and seclusion in British asylums.

It is the more desirable, because these facts are not always fairly represented. Thus, Dr. Gray, the editor of the *Journal of Insanity*, has lately returned from a visit to Europe, where, as he says, "he gave particular attention" to the subject of restraint. Since his return, at the suggestion of his Board of Managers that he should give them "a full presentation of the present status of professional opinion and practice on the question of restraint, whatever it be, fortified by such facts of experience as may throw light upon it *and furnish its justification*," he has come out with an elaborate defence of the use of mechanical restraint.

His own opinions given in the paper will have no more influence with thoughtful men, because he insists that, upon this subject, as well as the intimately correlated topic of employment for the insane, they have undergone no change during the last twenty years. Certainly, the light that has been thrown upon these subjects by British experience during that period, ought to have modified the views of every intelligent alienist.

He lays down three rules for the use of restraining apparatus.

"1st. *Cases of suicidal disposition where it is so determined and persistent that watchfulness will not secure the necessary safety.*

"2d. Where there is determined and persistent disposition to self-maiming or injury, or denuding the person, or debasing self-abuse.

"3d. *Where there is great destructiveness or violence toward others.*"

With the known characteristics of insanity we have in the above rules a warrant for a quite liberal use of mechanical restraint.

Dr. Gray uses the camisole, wristlets, the waist-belt, buckskin mittens, and, in rare instances, the leather muff. He makes no mention of the crib-bed, of which he has some thirty in number;

though in other asylums they are certainly used as means of restraint, if not at Utica.

The general conclusion of his paper is, that there is no real difference in principle among experienced professional men who have devoted their lives to this specialty; that the English Commissioners of Lunacy and the superintendents recognize the necessity of some mode of protective restraint; but having no settled convictions in favor of any particular method, they use coercive measures in the form of seclusion, the use of padded rooms, wet and dry packing, showering, and manual force of attendants.

The chaplain of the Utica Asylum has also, in a late number of the *Journal of Insanity*, attempted to show that non-restraint is a failure in England. The point upon which he lays most stress, perhaps, is the following:

"The report (referring to the Annual Lunacy Report) gives considerable attention to a review of cases of suicide in various institutions, in some of which deficiency of attendants is mentioned, but no reference is made, in connection with the circumstances related, *to the practicability of limiting these casualties by the judicious use of restraint.*" The thought is, that the English superintendents do not make what he calls a judicious use of restraint; that if they did, some of these suicides might have been avoided. The English statistics of restraint are as follows, taken from the Lunacy Report of 1880:

The county and borough asylums of England, which correspond in the main to our state asylums, are 59 in number. They contained 38,209 patients.

Twenty-nine of these, including an insane population of 17,756, or 46 per cent. of the whole number, used no mechanical restraint.

Eight with 5,057 inmates used neither restraint nor seclusion.

Eight with a population of 5,446 had, during the year, each but one occasion to use restraint, and that usually for surgical reasons.

In six others, with a population of 3,437, 27 patients were re-

strained by what is known as the "wet-pack" or "dry-pack." In these cases medical considerations prompted their use, as well as the purpose of restraint.

Of two asylums with 1,470 patients, the Commissioners of Lunacy make no mention of the use of restraint.

In fifteen asylums, with a population of 12,651 restraint, was used in the case of 115 persons. In a large number of these cases it is expressly stated that restraint was used for surgical reasons. But for any reason, in these fifteen asylums less than one per cent. of the persons were subjected to mechanical restraint.

Taking the aggregate population of all the county and borough asylums, less than four in a thousand ever had applied to them any form of mechanical restraint.

If we compare these statistics with the report of No. 17 asylum in Table 3, it will be seen that there is more restraint used in that single institution than in all the county and borough asylums of England.

And as the managers of the Utica Asylum have expressed a desire for light upon this question of restraint, we may add that counting the crib-bed as a form of mechanical restraint—and it most certainly is—there is more mechanical restraint used in their institution than in all the borough and county asylums of England. For, regarding all the 38,000 patients in such asylums as in one institution, the average number of persons under mechanical restraint, at any one time, would be but three.

As to seclusion, which means, according to the definition of the Lunacy Commissioners, putting the patient in a room by himself, usually with the door unlocked, out of 38,209 patients, only 911 were secluded. Of these, 47 were so secluded for bodily illness and not for excitement.

Deducting these and fourteen others, who for special reasons were secluded for protracted periods, the remaining 850 patients were each secluded, on an average, less than two days.

Following on the heels of this disuse of mechanical restraint have been other improvements in the same direction, which I have not space to describe.

Comparing the above statistics with those of the asylums in the United States, as seen in Table No. 3, and making allowance for the fact that the statistics in the one case are for a month, with an insane population of but 13,342, while in the other they are for a year, with a population of 38,209,—it will be seen that the difference in practice between the British and American superintendents, in the matter of restraint, is broader than the Atlantic.

But the alleged casualties are one of the bugbears of the non-restraint system. We have, unfortunately, no grounds for comparative statistics, for there is no public record of such events in American asylums. The English Lunacy Reports give publicity to all occurring in their asylums. The record for last year was as follows : With the 38,209 patients in county and borough asylums, more or less of them epileptics, paretics, and cases of senile dementia, 2 were scalded, 1 killed by another patient, 3 cases of broken ribs, one of which was through his own violence. I observe that these occurred principally in asylums where restraint was not wholly abandoned. I also observe that the list of such casualties is annually diminishing, in spite of the progress of the non-restraint principle.

There were eight suicides in these asylums. Here comparison is not entirely at fault. Thus, during the year 1875, I made some investigations and published the results, which have never been questioned. In the year 1875, in all the insane asylums of England, with a population of some 43,000 patients, there were but 21 suicides, or one to every 2,000 patients. During the same period, in 40 American asylums, containing 17,000 patients, there were 35 suicides, or one to every 500 patients. In other words, suicides were four times as common in American asylums as in those of England.

I may venture another comparison. The most pronounced opponents of the principle of non-restraint in this country are the superintendents of the asylums at Utica, and at Newburgh, Ohio.

In the county and borough asylums of England there were during the last year 4,291 deaths, and one suicide to every 536 deaths.

During the last 17 years, or as far back as my file of the Utica

Reports extends, there have been 951 deaths and 17 suicides, that is, one suicide to every 56 deaths ! !

At the Newburgh Asylum since its opening there have been 418 deaths and 15 suicides, or one suicide to every 28 deaths ! ! !

In conclusion, I think that it may be said—carrying the convictions of the reader—primarily, that "chemical restraint" is not the substitute for mechanical restraint in British asylums; incidentally, that the principle of non-restraint is not a failure in England; that casualties are not confined to non-restraint asylums; and lastly, that some of the advocates of mechanical restraint seem to be reluctant to have their methods made known to the profession generally. H. B. WILBUR.

Postscript.—The author of the above editorial article had intended not to name the various American asylums set down in his table, but had referred to them simply by numbers. I consider the subject one of such great importance to the medical profession, and to the public generally, that I have assumed the responsibility of re-inserting those names. This being done, persons interested in the more humane and intelligent care of the insane will know where to look for remnants of barbarous measures, for overdrugging, and for excessive suicide. E. C. SEGUIN.

NEW BOOKS AND INSTRUMENTS.

Photographic Illustrations of Cutaneous Syphilis. By George Henry Fox, A.M., M.D., etc. New York: E. B. Treat. 1881.

This atlas of photographic illustrations of cutaneous syphilis is intended by the author to be the complement of the "Atlas of Photographic Illustrations of Skin Diseases" lately published, and which has already been reviewed in these Archives. The plates number forty-three, and comprise seventy-three photographs. The "artotype" process has been employed, as in the other atlas; and the coloring has been applied by hand.

These plates represent all phases of cutaneous syphilis, from the initial lesion to the gummy tumor and ulcerative lesions of the latest stages of the disease, as well as a number of cutaneous affections not syphilitic, but which offer so many points of resemblance to certain syphilitic manifestations that a correct conception of them is of the greatest importance to the clinician; such, for instance, as condylomata acuminata, hydroa, eczema squamosum (palmare), scrofuloderma ulceratum, etc. The text accompanying the plates forms an essay upon cutaneous syphilis, the nature of the subject not allowing the author to follow out the plan pursued in the descriptive text of the Atlas of Skin Diseases.

In order to faithfully portray the features of a cutaneous eruption it is essential that the greatest possible attention should be devoted to the most minute details. This is not always the case in photographing pathological conditions. One can reproduce on paper, a fractured bone, or a deformity, or certain kinds of tumors, and the eye will derive correct impressions of essential features, whether the size of the object has been reduced or enlarged in the picture. The general effect is sufficient to produce the de-

sired impression. In cutaneous pathology it is quite otherwise; unless the portrait represents the lesion, even to its smallest detail, it is of no profit to the clinician, since in losing the resemblance to minutiæ, the distinctive features, and consequently the general resemblance, are likewise lost. The diagnosis of cutaneous diseases often depends upon the narrowest scrutiny of the lesion. If the artist would succeed in accurately picturing skin diseases, it is necessary that the copy should be of such a size as would admit of the illustration of all the characters of the eruption. Difficult as it often is to identify all the subtleties of skin lesions upon the living subject, it becomes impossible to do so in the illustration, when their size has been reduced to one fourth or one eighth that of the original. This is an error that was prominent in the Atlas of Skin Diseases, and is none the less noticeable in the atlas we are now considering. As examples of this we would especially refer to plates xiv, xxxv, and xxxviii, as presenting this fault in the highest degree; while, as exemplifying the much less difficult task of him, who, in producing portraits, would aim at general effect only, we would direct attention to the right-hand figure in plate xlvii, where the artist has given upon a surface no larger than one's thumb-nail the full-face representation of the distortion of the features by hereditary syphilis, that leaves nothing to be desired, and yet one looks in vain for the scar-tissue and other details that he knows must have been visible in the patient. It is impossible to show them upon so small a picture.

In considering the several plates it will be more profitable not to take them in regular order, but rather in groups illustrating the different forms and phases of eruption. It must be remembered, however, that a work of the kind before us is designed for the instruction of those whose opportunities for clinical observation are limited, as well as for those whose advantages enable them to become familiar with types of disease at the clinic, and that to the former class of observers the plates themselves must serve as the types not otherwise attainable. Deficiencies, imperfections, and errors are serious matters to the tyro, while the expert is able to unconsciously fill in the gaps and smooth down the asperities, and thus derive a benefit his less fortunate brother fails to receive.

Beginning with the initial lesion of syphilis we have plates xlii and xliii, representing chancre (infecting chancre), (two pictures). Accompanying these is one representation of chancroid for comparison, and in plate xliii a photograph of periadenitis. The in-

herent difficulties in picturing chancre are, doubtless, very great, nevertheless the author has succeeded in reproducing these lesions well, though the observer is left in doubt whether in plate xlii is represented a healing chancre or an "eroded chancre." Chancroid is moderately well shown. The size of these figures is so much reduced, however, that the eye fails to receive the impression that may be obtained at the clinic. Plates i, ii, iii, and iv show very successfully the erythematous syphiloderm and maculations succeeding it. They are, for the most part, admirable, and leave but little to be desired. We think the coloration a little too dark, and have to note here what was a decided defect in the Atlas of Skin Diseases, and what, we may say in passing, seems to be present throughout the whole of the present work, viz., differences in the intensity of color in similar photographs taken from different copies of the work. This is, indeed, a grave fault, but one, we believe, that is unavoidable where each picture has to receive the separate attention of the artist, and where, as is highly probable, it is not always the same hand that wields the brush. Pigmentatio post syphiloderma is perfect. Leucoderma post syphiloderma, shown in plate iii, is an example of a condition first described by the author, but which has yet not found, generally, the same interpretation as that given by him.

The papular syphiloderm and its varieties occupy a number of plates. The miliary and lenticular papular syphiloderms (plates v and vi) are excellent likenesses. In applying color, however, the artist has used black quite too liberally. The tiny punctæ seen all over the surface would afford excellent representations of comedo, and if seen upon the face would certainly be mistaken for such. We think Dr. Fox has been in error in introducing into the series plate viii. The figure is denominated "syphiloderma papulosa." The subject was a negro, in whom the eruption, a lenticular papular syphiloderm, is shown as being almost white in color. Such an eruption we have never seen, and, indeed, are aware of but a single recorded example of it (published some years ago by Dr. R. W. Taylor). We can hardly imagine a papular eruption of inflammatory origin white in color, primarily. We have only seen it feebly imitated when, in the negro, a lenticular papular eruption shows a loosening of the epidermis, a whitish color being given by the desquamating horny epidermis ; or when (still in the negro), the epidermis having been lost during an eruption, the new epidermis does not acquire its full pigmentation until a subsequent period. In the latter case we have seen

spots that by contrast do give a whitish appearance. At all events we think such a plate out of place in a collection of types. It belongs rather to the museum. Plate ix shows a rather unusual but highly interesting eruption, denominated syphiloderma papulosum circinatum. It is much like erythema multiforme (circinatum), as is well shown in the portrait. Syphiloderma papulosum humidum is excellently shown in plate xvii, especially in the right-hand figure, where one readily recognizes the peculiarities of the mucous patch. Condylomata lata and condylomata acuminata are displayed side by side in plate xliv, but the figures are entirely too small to be more than suggestive. Syphiloderma papulo-squamosum, like all scaly eruptions, offers almost insurmountable obstacles to the artist. We think the author has succeeded no better than others in much of this field.

A very interesting series, however, and one in which Dr. Fox has had far better success, is comprised in the plates devoted to syphiloderma papulo-squamosum of the palms. These are highly successful representations, four in number, in two plates. They are truly life-like (plates xviii and xix). Associated with these are two plates (xx and xxi) with four photographs : one of hydroa (phemphigus iris), a good likeness ; the other of eczema squamos. palmare. The plates of the earlier palmar syphiloderms show less resemblance to the eczema squamosum than do those of the scaly syphiloderm of the later stages of the disease, as seen upon the soles in plate xxiii, where, we think, it would be difficult, without the foot-note, to decide between them. This, however, is to be attributed not so much to faulty representation as to the close resemblances often existing.

There are four plates displaying the papulo-pustular and pustular syphiloderm. Of these, xiii is quite good ; xiv is also good, but loses much in the desire of the author to indicate the symmetrical distribution of the eruption, whereby is necessitated a much too great reduction in the size of the figure. In plates xi and xii we again observe especially the tendency to color the lesions too deeply, the areolæ being occasionally almost black. Here is also revealed, carelessness in the hand applying the color, where its hurry is shown in the dauby style whereby the pustular character of the eruption is indicated. In plate xv we do not observe sharply-defined characteristics, and are again reminded of comedo by the black dots scattered over the picture. (Syph. pustulos. corymbiforme.) The later pustular eruption, pustulo-crustaceous, is accurately represented in plate xxxi, but it appears

with less success in plate xxxii, where there is shown also a very unsatisfactory example of the tubercular syphiloderm.

Syphiloderma squamos. circinatum is another very successful example of palmar syphilis. Tubercular syphiloderm is shown, in various forms, in an interesting series of plates (tuberculo-crustaceum, tuberculo-squamosum, tuberculo-ulceratum, and tuberculo-serpiginosum). To these is appended, in plate xxix, a picture of scrofuloderma ulceratum, contrasting with them by its vivid coloration. Plate xxiv is a splendid illustration of tubercular syphiloderm, and will prove of the greatest value to the practitioner. Plate xxv has also a good delineation of the same lesion, but falls off in coloring. Plates xxvii, xxviii, xxix, and xxx show the tubercular, serpiginous, squamous, crustaceous, and ulcerative forms of late syphilis. These various lesions are well shown, especially in plates xxvii and xxix (where, in the former, the mingled cicatrization and ulceration of very late syphilis are exceedingly well depicted). Plate xxx of this series loses much in the attempt to put a large subject into too little space. Part of the coloring here also is so dark that the violet pigmentation of ecchymosis seems to have spread over the whole lower back. Onychia syphilitica is but imperfectly portrayed in plate xvi.

Syphiloderma gummatosum has received a large share of the author's attention. Plate xxxv, of this lesion, is without value on account of the too greatly reduced scale of the photograph; but plates xxxii, xxxiv, and xxxvi show this lesion to excellent advantage; indeed, we think the first-mentioned of these plates is nearly perfect.

The ulcerative syphiloderm is the subject of five plates. It is a very difficult task to delineate an ulcer, and yet in several of these Dr. Fox has met with gratifying success. Plate xl, for example, is a very good specimen. A decided drawback to the series, however, is the small size of the photograph; the ulcers are too diminutive, for the most part, to enable one to recognize the characters of their floors. The appearance of the granulations cannot be recognized, and the excavations that we know must be present are not made apparent. Plate xli shows very effectively the results of destructive ulceration of the nose.

The last four plates comprise photographs of lesions due to inherited syphilis. Dactylitis syphilitica appears, and as the striking results of the inflammation are easily portrayed the plate will give satisfaction though the photographs are misty. Plates xlv and xlvii illustrate the ravages of inherited syphilis upon the face, and

are most instructive productions. The picture of the child's face in xlv is invaluable, as showing the notched upper, central, permanent incisor teeth characteristic of this form of the disease, and as indicating the keratitis undoubtedly present. If the linear cicatrices so often seen around the mouths of those similarly affected could have been shown, the illustration might serve as the type of a large number of cases of inherited syphilis. Plate xlvi is a suggestive representation of ulcerative eruption from the inherited disease, but is too small to reveal the true features of the process. Periosteal thickening along the tibia is well displayed.

These plates, undoubtedly, possess many meritorious qualities. The knowledge that he is looking at the photograph of a real person impresses upon the observer the idea that he is examining the disease itself and not a mere "counterfeit presentment." He is thus enabled often to secure a better mental picture of the diseased condition than if he used some more perfect and accurate results obtained through other methods. It is evident that the real value of these plates depends upon the accuracy with which the coloring has been applied. Photography, so far as its capacity extends, *must* give a faithful portrait, provided the figure be reproduced in such proportions that it is possible to recognize the smallest visible details of the eruption in the picture. (We have already seen that the author has repeatedly fallen into the error of greatly reducing the size of the figure, one that much lessens the value of the work.) Unfortunately the coloration of the photographs can not receive the same scientific treatment, but is dependent upon the judgment, taste, and skill of an artist. In order to obtain uniform results each separate picture should receive, during its preparation, the personal attention of the author. This, in a work having an extensive sale, like the one before us, is manifestly impossible. Differences in degree of coloring must therefore occur, and one occasionally feels convinced that he who applied the color was more intent upon completing a given number of photographs than in securing an accurate resemblance to the original.

To return to a more agreeable portion of our task, we can say, in all conscience, that many of the plates present studies not only excellent in themselves, but which cannot be had in any other work with which we are acquainted, and which cannot fail to be of great value to the student, practitioner, and teacher. The deficiencies of the plates we think we have pointed out.

There can be no doubt that, had Dr. Fox given in the forty-eight plates, forty-eight photographs instead of seventy-three, but

more nearly approaching the size of life, both the Atlas and the medical profession would be gainers.

The text accompanying the plates is naturally addressed to the consideration of cutaneous syphilis, and is in every respect admirable. Beginning with a general introductory, the author, in the second chapter, considers "the examination of patients." This chapter is excellent and deserves the closest attention. The rules proposed for the guidance of the practitioner are most valuable. Chapter III discusses the stages and course of syphilis, which, in accordance with prevalent opinion, is considered as "a chronic exanthematous affection." The various periods of development, the stages, the course, etc., of the disease, receive due attention. The general characters of syphilitic affections are enumerated in the same admirable manner, and the modes of the transmission of syphilis are described at length. Next follows, in Chapter V, a description of the chancre in its several varieties, which the author thinks may be reduced to three clinical forms, viz. :

1. The dry papular chancre.
2. The eroded chancre.
3. The large indurated chancre.

Other forms of chancre represent but non-essential modifications of these. The confusion that is apt to arise from a more complicated classification is thus avoided. The description of these forms of chancre, with their modifications, is associated with a carefully compiled table of the comparative frequency with which chancre occurs upon the various parts of the body, both in males and females.

Chapter VII begins the study of the syphilodermata. A very interesting historical summary of the various classifications of these lesions follows. The author prefers to divide the syphilodermata into: "1. The early syphilides; and 2. The subsequent syphilides,"—the former class comprising the erythematous, papular, and pustular syphilides ; the latter, the tubercular, the squamous, the pustulo-crustaceous, the gummatous, and the ulcerative syphilides. The "early syphilides" are properly enough named, but we cannot regard the second class as happily designated, since the term "subsequent" is too indefinite and always presupposes that the observer has in mind the earlier lesions as standards of comparison. One would hardly feel comfortable in calling a gummy tumor "a subsequent syphilide" ; nor has this designation, as contrasted with "early," the same appropriateness

as the often employed and generally adopted terms "late" or "tardy" or even "tertiary" in describing the cutaneous lesions of late syphilis.

A large portion of the text is devoted to the consideration of these lesions, and we do not remember to have read a more accurate and more generally instructive account of their development, course, and terminations than is presented in these pages. The questions relating to the character and course of syphilis, and to matters of treatment, are discussed at length, and we regret that we cannot give a synopsis of this portion of the work. A valuable formulary is appended. [I. E. A.]

Artificial Anæsthesia and Anæsthetics. By HENRY M. LYMAN, A.M., M.D., Professor of Physiology and Diseases of the Nervous System in Rush Medical College, Chicago, Ill., etc. New York: Wm. Wood & Co., 1881, pp. 337.

This is the September volume of Wood's Medical Library of Standard Authors, and it alone would redeem many other of the inferior works which occasionally appear in this series. We do not mean to suggest that the work is faultless, but that it possesses a marked value. The subject hardly admits of much originality, as the author frankly confesses in his preface, wherein he states that he has quoted extensively from his predecessors in the field of artificial anæsthesia. What we would especially commend is the absence of that dogmatism which is so offensive and so unscientific. In explaining the *modus operandi* of anæsthetics the author impartially reviews the theories of others, and then in singularly clear language explains his own very satisfactory explanation of this abstruse subject. Throughout the whole book the facts are for the most part clearly and succinctly stated, although occasionally certain inelegancies and curious phrases strike the reader. We think, however, that the book is unnecessarily voluminous, owing to the introduction of extended sections on various substances which are practically useless as anæsthetics, and of details which more properly belong to a treatise on materia medica and therapeutics. Thus, several pages devoted to chloral hydrate used *not as an anæsthetic*, but as a remedy for delirium tremens, puerperal eclampsia, chorea, singultus, etc., seem to us misplaced. All these, however, are but minor defects not in any way detracting from the other undeniable merits of the work.

Dr. Lyman opens with a history of his subject, wherein he learnedly shows the antiquity of man's efforts to mitigate the pain

consequent upon operations. He shows that, as in the case of other great discoveries, artificial anæsthesia was not conceived of by any *one* man, but that isolated facts and experiments prepared the way, until, finally, many widely separated individuals—separated not only by space but time—independently discovered practical means and materials for the safe abolishment of pain. He then proves that in 1842 Dr. W. C. Long, of Jefferson, Ga., was actually the first who performed an operation under full anæsthesia, the agent used being ether. Other independent investigators followed, ignorant of Dr. Long's or each other's experiments. Finally, to Dr. T. G. Morton, who, on Oct. 17, 1846, first publicly anæsthetized a patient at the Massachusetts General Hospital, Dr. Lyman awards the palm of being the practical *introducer* —although not *discoverer*—of artificial anæsthesia. Further than this nothing need be said on the much-vexed historical point.

Next follows an elaborate consideration of the phenomena of anæsthesia in general, containing important facts which it were well if all administrators of ether, as well as chloroform, would read. We most heartily endorse what the author insists upon all through the work, that any anæsthetic is dangerous, and should only be confided to experienced hands.

After reviewing certain of the older theories as to the mode of action of anæsthetics, Dr. Lyman calls attention to the fact that various experimenters have demonstrated that oxidation is arrested in a very attenuated atmosphere of ether, chloroform, turpentine, and various other agents which *within* the body produce the phenomena of anæsthesia. Ether, when added to a fermenting solution of sugar, arrests the action, not by killing the fungus but by putting it to sleep, as it were, for, when the ether is removed by evaporation or otherwise, fermentation again actively proceeds. In like manner the natural processes of vegetable life are completely arrested by the vapor of ether, chloroform, etc., to be again healthily resumed upon their withdrawal. All these facts demonstrate that anæsthetics tend to place in abeyance molecular movement. According to the author further experiments show that certain "cell groups" of the organism succumb to this paresis of molecular movement sooner than others, which fact explains the progressive phenomena of artificial anæsthesia. These results are always dependent upon the actual contact of the paretic cells and the anæsthetic agent. The author next considers the phenomena of normal sleep, which present close analogies to those of anæsthesia. We wish that space permitted more ex-

tended quotations from this exceedingly lucid portion of the book, but we shall be compelled to allude to only such points as are absolutely essential for the proper understanding of our subject.

"Sleep commences in the organs of sensation. The sensory nerve tissues are evidently composed of matter in a condition of less stability than obtains in the other tissues of the body. If this were not the case sensation could never precede motion in the nerve centres. Every impulse that reaches a complex organ like the spinal cord or the brain must necessarily disturb the equilibrium of the more unstable molecules before it disturbs more stable masses. This unstable matter, therefore, constitutes a recipient apparatus for all impulses that move in a centripetal direction,—from the surfaces of the body to the central organs in the cerebro-spinal axis. While the constituent matter of the recipient apparatus preserves its condition of instability, it is said to be irritable, and by virtue of that irritability motion is liberated in its substance,—probably through rapidly successive isomeric changes,—and is transmitted to the specifically motor centres or to the apparatus of conscious sensation. But when, as a consequence of repeated changes of this nature, the irritable matter has deteriorated in nutrition, and has become overcharged with the waste products of tissue change, it is no longer the unstable substance it was at first. It no longer liberates motion under the influence of impulses from abroad. There can be no further distribution of motion through its agency, and the motor ganglia, with the organ of consciousness, can receive no excitement to action through the normal channels. During this condition of the recipient nervous apparatus the individual must necessarily remain ignorant of all that passes without. He becomes unconscious of the external world; he may even lose all consciousness of self; we say that he is asleep. This condition may be the result of the ordinary tissue changes that conform to the daily rotation of the earth, and then the sleep is natural; or it may be a pathological result of morbid conditions of the body, constituting a condition of stupor; or, finally, it may result from the presence and peculiar energy of certain substances that have been artificially introduced into the blood, producing artificial anæsthesia. The causes differ but the effect on consciousness is the same."

The author then cites the experiments of Durham, Bedford Brown, and others, which conclusively show that the conditions

of the circulation, etc., are similar in both sleep and anæsthesia. Fortunately for the success of anæsthesia, the respiratory nerve centre retains its irritability longest, and is the "ultimum moriens" in cases of death from anæsthetics. The author concludes that "molecular paralysis, then, is the cause of all the varied phenomena of anæsthesia," and that this is not due to any chemical combination with the tissues. Even the excitation evident at the outset of anæsthesia from ether is not properly its *primary* effect, for in reality the first effect of the ether vapor is a *paralysis of the vaso-motor* nerves, which induces relaxation first of the pulmonary then of the cardiac—coronary—vessels after a slight preliminary narrowing of their calibre. From the increased blood supply of the heart it acts more rapidly,—more blood passes through the brain and other capillaries in a given time,—whence results a temporary increase of every function, but especially that of the cerebro-spinal nervous system.

The author explains the apparent primary irritant effect on the blood-vessels, as evidenced by their contraction, by showing that it is really due to the paralysis of the centripetal nerves connected with a central motor ganglion, which liberates motion by disturbing the equilibrium usually existing "between their protoplasm and the protoplasm of the structure by the aid of which such liberation is ordinarily initiated." The primary contraction of the blood-vessels explains the increased vascular pressure observed at the outset of anæsthesia. The acceleration of respiration is due to increased vascularity of the nerve centres. The free secretion of saliva and mucus has been shown by experiment to be chiefly dependent on the local action of the anæsthetic vapor, which also excites reflex manifestations of cough. Exalted sensibility of the sensorium, with exaggerated muscular contractility, is due to the unusual blood supply of the nerve centres, which perhaps explains the fact that certain painful operations *commenced* before the induction of full chloroform anæsthesia have *at once* terminated fatally from the increased perceptivity of the cerebro-spinal axis.

In describing the mode of administering anæsthetics the author insists upon a physical examination of the patient, and the giving of due weight to any passing illness or excessive mental excitement, which *temporarily* depresses the vital powers, and which may be usually overcome, in the latter event, by tact, aided, perhaps, by a little alcoholic stimulus. The rule of abstinence from solid food for a few hours before the induction of anæsthesia—

not for many hours, thus inducing exhaustion—especially in the case of ether, should, whenever possible, be rigidly enforced. "The administration of the anæsthetic vapor should never be confided, excepting in special emergencies, to unprofessional or unskilful hands."

Accidents are indeed rare, but they might occur less frequently if this simple rule was observed. We have more than once seen alarming asphyxia, induced by careless etherization, which we are convinced would have proved fatal, unless averted by the interference of an experienced by-stander, whose advice has been more than once strenuously opposed ! Dr. Lyman advocates, except for nitrous oxide gas, that the anæsthetic vapor should be administered in a dilute form *at first.* The temporary removal of the inhaler if marked laryngeal spasm occurs is inculcated. Careful administration of the anæsthetic under such circumstances is called for, instead of the hurried forcing of the inhalation, which is *always* dangerous, as such cases belong "to that class in which the transition from the excitement of delirium to the collapse of syncope is so often sudden and dangerous." In common with the author we think the plan of pressing the inhalation to check incipient vomiting is dangerous.

Dr. Lyman condemns all the complicated forms of inhalers, which, although theoretically perfect, are practically dangerous. They do not effect a uniform dilution of the anæsthetic vapor, and, besides, "a definite dilution of the more powerful anæsthetics is no safeguard against danger in their use. A dose which produces no appreciable harm in thousands of cases will promptly destroy the life of a susceptible patient. It is not so much the excessive percentage of vapor that kills, as *it is the excess of vapor without regard to percentage.*" * * * "The varying conditions of the same patient, as regards his health or vigor, exercise a great effect." * * * "Consequently a person may succumb to-day in the presence of a dose which he has on previous occasions received without the slightest risk." * * * "It is by reference to the quantity of the anæsthetic substance in contact with the tissues of the body that the process of inhalation must be regulated." "The operator (anæsthetizer) should let nothing intervene between himself and the patient." The author of course figures and describes the best-known forms of inhalers, but we are surprised that he has not mentioned that of Dr. O. H. Allis, of Philadelphia, which is open to no objections such as Dr. Lyman has so justly urged against other forms of appa-

ratus, as it aims neither to regulate the access of air nor the percentage of anæsthetic vapor, being merely a cleanly, cheap, and *economical* apparatus. Codman and Shurtleff's mouth-pieces for the inhalation of nitrous oxide gas are recommended as the best.

In treating of the accidents of anæsthesia the author only deals with "those alarming symptoms of cessation of life" which "have supervened during the administration of the anæsthetic, or before the normal conclusion of the period of anæsthesia." The administrator should always remember that since "the phenomena of anæsthesia are the result of a paralyzing action," every "inhalation is a step in the direction of respiratory paralysis and death." Any thing which diminishes the functional power of the lungs or heart, such as cold, privation, mental anxiety, loss of sleep or blood, alcoholic intoxication, injury of the respiratory centres from "shock,"—which explains the wisdom, at least in *civil* surgery, of waiting for the subsidence of this condition,—lung diseases by their increased liability to syncope, organic disease of the heart or great vessels, are always dangerous and are generally, according to the author, counter-indications to anæsthesia. We consider this is too sweeping an assertion, although such circumstances demand great caution in the administration of anæsthetics. Pain, when the receptive centres are in an exalted state of sensibility before the induction of complete anæsthesia, seems quite often to have been a cause of death. "Excitement and fear may overpower the heart to a degree that shall constitute all the difference between safety and danger." Although the foregoing remarks apply chiefly to chlorororm and the more powerful anæsthetics, it must be remembered that as death has occurred from the use of ether they are to a degree applicable to this also.

The two principal dangers of anæsthesia are cessation of respiration and circulation. Restoration of the former function should be *the* aim of the administrator, in the event of the appearance of dangerous symptoms, and the efforts should be prolonged and *systematic.* The author places *inversion* of the patient in the foremost place, instancing various cases in proof of this position. Sylvester's or Howard's method of effecting artificial respiration is next in importance. Drawing forward the tongue only acts as any other peripheral irritation does. We are surprised that the author omits the really efficient plan of grasping the lower jaw on each side, and by the fingers *depressing* the symphysis while the thumbs *forcibly* press forward and elevate the angles. I

have repeatedly practised this manœuvre, instantly relieving the stertorous respiration, which is again resumed as soon as the hands are removed. In consequence, such a position of the jaw should be maintained until normal respiration returns. The forcible dragging forward of the tongue is often absolutely harmful, as it arrests the natural play of the larynx during respiration. Faradization of the phrenic nerves is theoretically useful, but "should be combined with the attempt to fill the lungs by Howard's method." "One electrode should be forcibly pressed over the right phrenic in the neck," * * * "while the other electrode should be placed" * * * "over the sixth intercostal space on the right side," * * * "and these applications should be renewed about twenty times a minute." The *left* phrenic should never be faradized to avert impending syncope, since, as the author states, Richardson has shown by actual experiments on animals that the passage of an electric current *through* the heart during anæsthesia with failing heart action "*will at once arrest all movement.*" The current "should never be very energetic." General electrization of the surface and electro-puncture of the heart are worse than useless from the risk of tetanizing the feebly pulsating heart.

If artificial respiration and electricity be not available, or tracheal catheterism fail in introducing air into the lungs, tracheotomy and tracheal insufflation should at once be tried. In view of the importance of the subject no apology is needed for giving Dr. Lyman's own summary of rules.

"In every instance of syncope, threatening or actual, the head should be depressed to the utmost limit of bodily inversion. Artificial respiration, preferably by the aid of rythmical pressure upon the chest and abdomen, should be commenced at once, and should be continued, deliberately and persistently, until the patient is either out of danger or is unquestionably beyond all hope of recovery. While these artificial movements are effected" * * * "an assistant should attempt the introduction of the laryngeal sound" (catheter ?) "in order to supplement the thoracic movements by laryngeal insufflation. Failing in this endeavor, tracheotomy should be performed, and the sound should be introduced into the windpipe through the wound in the neck." Anæsthetic mixtures are evidently not highly thought of by the author, and he thinks that the bulk and complexity of the apparatus for the precedent use of nitrous oxide before ether inhalation, although in many ways unobjectionable, will always restrict its

use. The mixture of ether vapor and nitrous oxide is also *explosive.* The weight of opinion seems against the previous administration of chloral and morphia, although Koenig thinks it useful in hard drinkers by considerably lessening the period of excitement under chloroform. In midwifery the author gives much sound advice as to the *mitigation*, not abolishment, of the pain, etc., and points out that deaths have occurred even during labor, so that cardiac and respiratory weakness in such cases are positive counter-indications.

Dr. Lyman thinks that chloroform *should never be used in dentistry*, but when administered it should be carried to full anæsthesia, lest in certain cases the shock of pain fatally overwhelm the already feebly beating heart. The remarks on local anæsthesia are good, but we are surprised to note that one of its most troublesome after-results is not mentioned, viz., the free oozing due to vaso-motor paralysis, which we have always failed to check, except by firmly applied pressure. This is sometimes difficult and painful to maintain. Owing to the impossibility of collecting any thing like *all* the cases where artificial anæsthesia has been induced, Dr. Lyman considers his figures as only approximately correct, and thinks—in fact, has good reason to know—that many more deaths have occurred than the general profession is aware of. His figures are: For ether, 99,255 inhalations, 6 deaths—1:16,542 ; for chloroform, 492,235 inhalations, 84 deaths—1:5,860.

The section on the "Medico-Legal Relations of Anæsthesia" is of great interest, but space fails for more than the statement that a sufficient number of well-authenticated cases are adduced to prove the following proposition : "That chloroform may be administered during sleep in such a way as to facilitate the commission of crime, but that this result is not likely to happen in ordinary unskilled use of the anæsthetic."

We shall now endeavor to place before our readers the most striking and important facts relative to the various anæsthetics brought out by our author, although much valuable and interesting matter must necessarily be omitted for want of space.

As to methylene bichloride the author sums up as follows : "Taking, therefore, into consideration the physical properties of the liquid, the inflammability of its vapor, its considerable expense, and the absence of any special advantages over chloroform, together with the high rate of mortality that has attended its use, it is impossible to recommend the further employment of bichloride of methylene." Its action is very similar to that of

chloroform. The author calls attention to the fact that any impurity in chloroform, such as alcohol, may produce distressing and dangerous symptoms, and that it does not always follow that the label of a prominent manufacturing chemist ensures perfect purity. Under ordinary circumstances death occurs from the gradual overwhelming of the centres of respiration and circulation *plus* a positively poisonous effect upon the mechanism of the heart itself. Sudden death, on the other hand, is due to violent irritation of the terminal branches of the pneumogastrics in the lungs, which is reflected from their centres and produces "inhibitory arrest" of the heart "in diastole." Chloroform may be eliminated by the mammary glands, as well as by the way of the lungs, skin, and kidneys. The urine may give a decided reaction to the copper test, which, however, is not conclusive either of the presence of chloroform or sugar, since the alkaline formiates "also reduce the cupro-potassic solution." Chloroform inhalation is always dangerous "to the victims of chronic alcoholism." The author calls attention to a useful application of chloroform vapor, viz., to the destruction of the larvæ of flies, *i. e.*, "maggots," in the nasal passages or exposed wounds. The author graphically demonstrates, by a series of sphygmograms, the depressing effect of chloroform on the heart, both of the lower animals and man. According to the Committee of the London Medico-Chirurgical Society "the depth of respiration became less and less, and after the stage of perfect insensibility was reached the amount of air entering the chest was exceedingly small." All administrators should remember that any percentage of chloroform in the air beyond 5 per cent. is dangerous, and that in consequence the effects of each addition of the liquid in the inhaler or on the napkin must be jealously watched. Dr. Lyman prefers a few folds of lint for the administration of this agent. There is a decided lowering of temperature, which is usually in proportion to the duration of anæsthesia, and commonly reaches its maximum long after the cessation of anæsthesia. In a review of the details of the 393 fatal cases of chloroform anæsthesia the reader cannot but be struck with its great danger, since in numerous cases death has occurred in *less than three minutes—even under a minute*—where a *very* small portion of the drug was used.

Space will not admit of our giving the various interesting facts contained in the summary with which the author closes the section on chloroform anæsthesia, except that he thinks he has demonstrated that "the actual structural condition of the heart must

necessarily occupy a much less important position among the causes of death under chloroform than was once supposed." From its slighter effect upon the heart ethydene dichloride, although not without danger, is considered to be decidedly better than chloroform. Cardiac syncope seems the main source of danger. It is demonstrated by means of numerous sphygmograms that ethylic bromide produces much greater cardiac depression than ether, the pulse curve being nearly identical with that of chloroform. The bromic impurities apt to be present in this liquid are more dangerous than the impurities of chloroform. "Altogether, it must be admitted that ethylic bromide is to be ranked with chloroform as one of the most potent and most dangerous of anæsthetic substances." We cannot agree with the author as to the efficiency or advisability of carbolic acid as a local anæsthetic, but we certainly commend him for calling attention to the risks of poisoning from carbolized dressings, which, however, is of very rare occurrence. Chloral hydrate as an agent for the production of surgical anæsthesia *may* be of occasional use when administered by the mouth to children, but the intravenous method "possesses all the risks that attend the inhalation of chloroform, and is characterized by many additional inconveniences and dangers," which the author sets forth at length. The experiments of the London committee before mentioned demonstrated in the clearest manner the superiority of ether over any other anæsthetic. Death in the animals experimented upon occurred from failure of respiration. This seemed to be induced partly from paralysis of the respiratory centres, partly from the local effect on the pulmonary tissues. Identical changes in the lungs were produced by ether, chloroform, and ethydene dichloride. We quote some interesting results obtained by this committee: "When anæsthetics are administered in excessive quantities the first change noticed in the circulation in the lung is a diminution in the rapidity of the flow in the capillaries; and this, notwithstanding that the number of the heart's impulses remains unchanged and the circulation through the larger vessels is unimpaired. Very shortly after this, instead of the flow of blood being constant, it gradually becomes intermittent—first in the capillaries, afterward in the arterioles, and subsequently in the larger vessels. This intermission in the flow of blood is followed by a swinging to-and-fro movement of the corpuscles just previously to the stoppage of the circulation through the capillaries. It must now be observed that the stoppage of the circulation in the lung takes

place first in the capillaries, then in the arterioles, and, last of all, in the larger vessels ; further, that the sequence in recovery is exactly the reverse. Again, it is to be noticed that the circulation in the foot stops not previously to, but shortly after, that of the lung ; and its re-establishment never occurs before, but always subsequently to, the restoration of the pulmonary circulation."

These phenomena are partly due to "impairment of cardiac vigor," partly to a "local resistance to circulation," the result of a change of relationship between the blood corpuscles and the capillary walls. Sphygmograms show that under ether, "during the most profound insensibility, the ascending branch of the curve maintains its original angle with the perpendicular, showing that the ventricular contractions have lost none of their relative vigor." When death occurs from the administration of ether, "respiration almost invariably ceases before the heart is arrested." The bodily temperature is reduced in all cases of ether anæsthesia, the maximum being 1.5° C. Good advice is given as to abstinence from solid food for some time before inhalation, thus lessening one of its risks, viz., suffocation from passage of fragments of food into the trachea during vomiting.

The author adduces several cases of severe neuralgia occurring after ether anæsthesia, but as all the patients were women it is possible that although *post hoc* it was not *propter hoc*. A caution is given about the inflammability of ether, which, although commonly known, is too often ignored, as accidents testify. As to the dangers of anæsthesia by ether the author points out that although *slight* they are *real*, and that whenever the pulse grows feeble and the face pale, while respiration assumes an irregular and superficial character," that there is danger, and that the inhalation should be suspended. He distinctly calls attention to the most common source of danger in inexperienced hands, viz., that of asphyxia. We have over and over again seen a dangerous degree of asphyxia produced not by too much ether, but by too little air mingled with the ether. Forcing forward the angles of the jaws, as before directed, and the admission of fresh air usually at once relieve the livid appearance and stertorous respiration, but there should very seldom be any necessity for a resort to this measure if the agent is carefully administered.

Intestinal obstruction holds the first place as to rendering ether unsafe, malignant neoplasms come next, while in tetanus and delirium tremens it is probably "quite as dangerous as chloroform." Any thing like old age, or some accidental condition producing

exhaustion, especially one likely to interfere with the function of respiration, renders the administration of ether more or less perilous. Death is usually the result of some kind of asphyxia. Sometimes vomited matters enter the larynx, at others the patient drowns in his own excessive bronchial secretions, but, as a rule, death "results from direct paralysis of the respiratory centres," the heart action persisting for some time after respiration has ceased. Chloroform, on the other hand, overwhelms the heart almost at the same instant that it paralyzes the respiratory centres.

In addition to sudden death from the direct effect of ether, in certain rare instances, "after the immediately anæsthetic effects have disappeared, the patient may become delirious, or comatose, or suddenly asphyxiated, and the case may result fatally a number of hours after the operation,"

Amylic nitrite is considered *in extenso*, but, it seems to us, very unnecessarily, as it certainly cannot be used as a surgical anæsthetic; but "its powerfully exciting effect on the heart, and the dilatation of the cerebral vessels that follows its use," certainly have served "to counteract the depressing effects of chloroform upon the circulation" in a number of instances. A few inspirations of its vapor usually suffice. If respiration has ceased hypodermic injection is recommended, but certainly great caution must be exercised when thus administered, since amylic nitrite is itself a powerful depressing poison. For certain *brief* operations demanding chloroform a mixture of ʒ ij of amylic nitrite to one pound of chloroform seems to have acted well, since when inhaled the face becomes flushed instead of pale, and the heart acts strongly. Some fifteen or more pages, illustrated by sphygmograms, are devoted to an excellent dissertation upon nitrous oxide. We shall merely give the conclusions arrived at by the author.

"1. Nitrous oxide gas possesses special anæsthetic properties.

2. If inhaled without dilution it produces asphyxia, as well as anæsthesia, by exclusion of oxygen from the blood.

3. Nitrous oxide does not enter into any chemical combination with the elements of the body, but is simply dissolved in the blood; hence its speedy entrance and departure from the organism.

4. Nitrous oxide is not decomposed in the blood, consequently it cannot replace oxygen or yield oxygen for the respiration of the tissues.

5. Nitrous oxide produces special effects upon the nervous system. When diluted with air these effects are limited to the

manifestation of a peculiar exhilaration. When inhaled without dilution, the gas produces first excitement, then anæsthesia, and finally asphyxia. The ultimate phenomena of asphyxia (convulsions, etc.) are suppressed by the anæsthetic energy of the gas.

6. In order to saturate the system with nitrous oxide sufficient for the production of anæsthesia the tension of the gas must equal the pressure of the atmosphere. A mixture of the gas with oxygen or common air will therefore produce complete anæsthesia, if it be inhaled under a pressure sufficient to raise the tension of the gas in the mixture to an equivalent of the tension of the undiluted gas under the normal atmospheric pressure. By this method anæsthesia and normal respiration may be indefinitely prolonged with perfect safety."

This last "conclusion" contains the gist of Paul Bert's experiments, and explains his system of administering the gas which has proved eminently successful, but owing to the cumbrous nature and expensiveness of the requisite air-tight chamber its use must, at least for the present, remain limited. Death has occurred during the administration of this apparently absolutely safe anæsthetic, and in rare instances unpleasant after-effects have been complained of.

A page is devoted to the consideration of anæsthesia by rapid respiration, with an attempt, differing from that of its author, Dr. W. G. A. Bonwill, the Philadelphia dentist, to explain the rationale of its action. We can testify to its efficacy as an almost complete annihilator of pain. We have had a crucial experiment tried upon our own person with such a diminution, nay, almost *complete* abolishment of pain, as to render tooth-drawing no longer a terror. Doubtless some persons are more susceptible than others. In our own person numbness was marked up to the elbow, more or less so all over the body, with, during most of a prolonged operation, nearly complete insensibility to pain in the mouth. Consciousness and *tactile* sensation are but little affected. To be effectual the author's directions must be strictly followed. Where successful it provides us with a perfectly safe, always available anæsthetic for operations not exceeding thirty seconds. For details we must refer our readers to the *Scientific American Supplement*, No. 275, April 9, 1881. In all, 48 substances, possessing in varying degrees the power of producing anæsthesia, are treated of by Dr. Lyman. Much valuable information is given, which we regret being unable to comment upon. Enough has been said to show how valuable the book is, and that any one would do well for the sake of this

one work to subscribe to Wood's Library. We congratulate Dr. Lyman on having added something worth reading to our medical literature, and are pleased to have this earnest of what is to be expected of the section on anæsthesia in the forthcoming International Encyclopædia of Surgery which will be written by him.

[C. B. N.]

A Manual of Practical Normal Histology. By T. MITCHELL PRUDDEN, M.D., Director of the Physiological and Pathological Laboratory of the Alumni Association of the College of Physicians and Surgeons, New York, etc., pp. 265, G. P. Putnam's Sons, 1881.

Owing to the general recognition of the importance of practical instruction in the methods of laboratory work, and the necessity of becoming familiar with the structure and appearances of the tissues and organs of the body, histological manuals have, of late, multipled with great rapidity. One of the latest contributions to this department is Prudden's Manual, and its author gives evidence of familiarity with and proficiency in his work, and evinces an intimate knowledge of the wants of students only to be acquired through years of actual teaching. The little volume also shows care and painstaking in its preparation.

It is eminently a practical manual, valuable alike to both student and practitioner. The method employed in the work is admirable; a brief description of each tissue or organ is given, followed by an account of the way in which the structures described may be demonstrated. The explanations are clear and concise, and no serious difficulty ought to be encountered in verifying them.

The chapters upon connective tissue and the members of the connective-tissue group are especially good, and should prepare the mind of the student for the intelligent understanding of the blending of the warp and woof of the simple tissues in the construction of more complex organs.

This little work will be found valuable in the hands of workers in the laboratories of our medical schools. Slowly the colleges of our land are swinging into line, and ere long they will demand of their graduates a practical knowledge of these important subjects, and we hope that the day is not far distant when all such institutions shall be required to include it in the college curriculum.

One source of regret with this volume, however, is the ab-

sence of all plates or illustrations in the text. A number of original drawings executed by so thorough and competent an observer could not fail to contribute additional value to the work, and we trust that a later edition may contain this desired improvement. [W. H.]

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